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Set Items Description

S1 499 ((INTERLEUKIN OR IL)(W)(1 OR 1A OR 1B OR 1ALPHA OR 1BETA OR IA OR IB OR IALPHA OR IBETA OR IRN OR 1RN) OR IL1 OR IL1A? OR IL1A OR IL1B? OR IL1B? OR IL1RN OR IL1RN) AND (MENOPAUS? OR PERIMENOPAUS? OR PREMENOPAUS? OR CHANGE(1W)LIFE)

S2 53 S1 AND (MUTAGEN? OR MUTAT? OR MUTANT? ? OR POLYMORPH? OR P-OLY(W)(MORPHISM? ? OR MORPHIC?) OR (VARIAT? OR VARIANT? ?)(5N-)ALLEL?)

S3 50 RD (unique items)

>>>No matching display code(s) found in file(s): 65, 129, 158, 624

3/3,AB/1 (Item 1 from file: 149)
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-key terms

02085035 SUPPLIER NUMBER: 87347833 (USE FORMAT 7 OR 9 FOR FULL TEXT)
Announcing the American Diabetes Association's 62nd scientific sessions.
Diabetes, 51, 6, NA(68)
June,
2002
PUBLICATION FORMAT: Magazine/Journal; Refereed ISSN: 0012-1797
LANGUAGE: English RECORD TYPE: Fulltext TARGET AUDIENCE: Professional
WORD COUNT: 15380 LINE COUNT: 03398

3/3,AB/2 (Item 2 from file: 149)
DIALOG(R)File 149:TGG Health&Wellness DB(SM)

02079894 SUPPLIER NUMBER: 86035147 (USE FORMAT 7 OR 9 FOR FULL TEXT)
Familial expansile osteolysis (excessive RANK effect) in a 5-generation American kindred.
Whyte, Michael P.; Reinus, William R.; Podgornik, Michelle N.; Mills, Barbara G.
Medicine, 81, 2, 101(21)
March,
2002
PUBLICATION FORMAT: Magazine/Journal; Refereed ISSN: 0025-7974
LANGUAGE: English RECORD TYPE: Fulltext TARGET AUDIENCE: Professional
WORD COUNT: 14115 LINE COUNT: 01149

3/3,AB/3 (Item 3 from file: 149)
DIALOG(R)File 149:TGG Health&Wellness DB(SM)

02072288 SUPPLIER NUMBER: 84666748 (USE FORMAT 7 OR 9 FOR FULL TEXT)
Interaction of hemostatic genetics with hormone therapy *; new insights to explain arterial thrombosis in postmenopausal women.
Braunstein, Joel B.; Kershner, Dawn Warner; Bray, Paul; Gerstenblith, Gary; Schulman, Steven P.; Post, Wendy S.; Blumenthal, Roger S.
Chest, 121, 3, 906(15)
March,
2002
PUBLICATION FORMAT: Magazine/Journal; Refereed ISSN: 0012-3692
LANGUAGE: English RECORD TYPE: Fulltext TARGET AUDIENCE: Professional
WORD COUNT: 11642 LINE COUNT: 01053

3/3,AB/4 (Item 4 from file: 149)
DIALOG(R)File 149:TGG Health&Wellness DB(SM)

01922506 SUPPLIER NUMBER: 63713371 (USE FORMAT 7 OR 9 FOR FULL TEXT)
Periodontal disease.(ABC of Oral Health)
Coventry, John; Griffiths, Gareth; Scully, Crispian; Tonetti, Maurizio
British Medical Journal, 321, 7252, 36
July 1,
2000
PUBLICATION FORMAT: Magazine/Journal; Refereed ISSN: 0959-8146
LANGUAGE: English RECORD TYPE: Fulltext; Abstract TARGET AUDIENCE: Professional
WORD COUNT: 2257 LINE COUNT: 00209

ABSTRACT: Periodontal disease is a disease of the gums. It is usually caused by an accumulation of plaque on the teeth. The plaque irritates the gums, causing gingivitis. About 90% of the population has gingivitis to a certain degree, which manifests as bleeding during tooth brushing. Unless plaque is removed by tooth brushing and flossing, the condition will progress to periodontal disease. A pocket forms around teeth, and the erosion of bone will cause the tooth to fall out eventually. Proper dental hygiene is essential for preventing periodontal disease.

3/3,AB/5 (Item 5 from file: 149)
DIALOG(R)File 149:TGG Health&Wellness DB(SM)
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01919150 SUPPLIER NUMBER: 63255073 (USE FORMAT 7 OR 9 FOR FULL TEXT)
Genetic factors and osteoporotic fractures in elderly people.(Letter to the Editor)
MacGregor, Alex J; Snieder, Harold; Spector, Tim D; Nemetz, Andrea; Pens, Amado Salvador; Kannus, Pekka; Kaprio, Jaakko; Koskenvuo, Markku; Palvanen, Mike; Parkkari, Jari
British Medical Journal, 320, 7250, 1669
June 17,
2000
DOCUMENT TYPE: Letter to the Editor PUBLICATION FORMAT: Magazine/Journal; Refereed ISSN: 0959-8146 LANGUAGE: English RECORD TYPE: Fulltext
TARGET AUDIENCE: Professional
WORD COUNT: 1860 LINE COUNT: 00172

3/3,AB/6 (Item 6 from file: 149)
DIALOG(R)File 149:TGG Health&Wellness DB(SM)
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01900246 SUPPLIER NUMBER: 61635220 (USE FORMAT 7 OR 9 FOR FULL TEXT)
Endothelial Apoptosis(*).
Stefanec, Tihomir
Chest, 117, 3, 841
March,
2000
PUBLICATION FORMAT: Magazine/Journal; Refereed ISSN: 0012-3692
LANGUAGE: English RECORD TYPE: Fulltext TARGET AUDIENCE: Professional
WORD COUNT: 10929 LINE COUNT: 01023

3/3,AB/7 (Item 7 from file: 149)
DIALOG(R)File 149:TGG Health&Wellness DB(SM)
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01890422 SUPPLIER NUMBER: 59961956 (USE FORMAT 7 OR 9 FOR FULL TEXT)
Women and Tobacco: Oral Health Issues.
Fried, Jacquelyn L.
Journal of Dental Hygiene, 74, 1, 49
Wntr,
2000
PUBLICATION FORMAT: Magazine/Journal; Refereed ISSN: 1043-254X
LANGUAGE: English RECORD TYPE: Fulltext; Abstract TARGET AUDIENCE: Professional

WORD COUNT: 5578 LINE COUNT: 00474

AUTHOR ABSTRACT: As a female-dominated profession, dental hygiene has a heightened interest in women's health issues. An area of disease prevention and health promotion that merits gender specific interventions is tobacco use. Tobacco initiation, habituation, and cessation are different for men and women, and their effects on women's health also are more varied and unique. This paper addresses trends in tobacco use by women, gender specific developmental and sociocultural considerations in initiation and habituation; the pregnant tobacco user and the developing fetus; *menopause"**, osteoporosis, heart disease, and tobacco use; the economic impact of pregnancy and tobacco use; and successful interventions with women who use tobacco. Since women present for more dental office visits than males, female patients are more accessible to the dental hygienist's tobacco intervention message, and gender specific strategies may be most successful. Dental hygienists' strong counseling and motivational abilities, along with their inherent interest in women's issues, can make prevention and cessation activities with female patients who use tobacco both challenging and rewarding.

Key Words: Tobacco, pregnancy, tooth development, health promotion.

3/3,AB/8 (Item 8 from file: 149)
DIALOG(R)File 149:TGG Health&Wellness DB(SM)

01889341 SUPPLIER NUMBER: 59643741 (USE FORMAT 7 OR 9 FOR FULL TEXT)
Gene Therapy in Orthopaedics.
Evans, Christopher H.; Robbins, Paul D.
Orthopaedic Nursing, 19, 1, 16
Jan,
2000
PUBLICATION FORMAT: Magazine/Journal; Refereed ISSN: 0744-6020
LANGUAGE: English RECORD TYPE: Fulltext; Abstract TARGET AUDIENCE: Professional
WORD COUNT: 3647 LINE COUNT: 00316

AUTHOR ABSTRACT: Genes are composed of deoxyribonucleic acid (DNA), the hereditary material of all nucleated cells. One way in which genes function is to direct the synthesis of specific proteins. When a gene is transferred to and expressed within a cell, the recipient cell produces the protein encoded by the transferred gene. This process forms the basis for gene therapy, which can be defined as the transfer of genes to patients for therapeutic purposes. Both genetic and acquired disorders may be treated, or even cured, by gene therapy. Potential orthopaedic applications include the treatment of arthritis, tumors, osteoporosis, and genetic diseases such as osteogenesis imperfecta, as well as the enhancement of tissue repair and regeneration. Impressive preclinical progress has been made in several of these areas. A phase I clinical trial of gene therapy for rheumatoid arthritis has just been completed. Orthopaedic gene therapy should become a clinical reality during the next decade.

3/3,AB/9 (Item 9 from file: 149)
DIALOG(R)File 149:TGG Health&Wellness DB(SM)

01887253 SUPPLIER NUMBER: 59579111 (USE FORMAT 7 OR 9 FOR FULL TEXT)

Influence of vitamin D deficiency and vitamin D receptor *polymorphisms"** on tuberculosis among Gujarati Asians in west London: a case-control study.(Statistical Data Included)
Wilkinson, Robert J; Llewelyn, Martin; Toossi, Zahra; Patel, Punita; Pasvol, Geoffrey; Lalvani, Ajit; Wright, Dennis; Latif, Mohammed; Davidson, Robert N
The Lancet, 355, 9204, 618
Feb 19,
2000
DOCUMENT TYPE: Statistical Data Included PUBLICATION FORMAT: Magazine/Journal; Refereed ISSN: 0099-5355 LANGUAGE: English
RECORD TYPE: Fulltext; Abstract TARGET AUDIENCE: Professional
WORD COUNT: 4089 LINE COUNT: 00391

ABSTRACT: Vitamin D deficiency may contribute to the high occurrence of tuberculosis in a population of Gujarati Asians living in London. Susceptibility to disease from Mycobacterium tuberculosis infection is influenced by environmental and host genetic factors, and vitamin D metabolism restricts its growth. Of 126 untreated patients with tuberculosis and 116 unrelated people without symptoms but who had contacted and been sensitized to tuberculosis, active vitamin D metabolism was recorded in 103 patients and 42 contacts. The levels were low in all patients and significantly lower or completely undetectable in the patients who had tuberculosis.

3/3,AB/10 (Item 10 from file: 149)
DIALOG(R)File 149:TGG Health&Wellness DB(SM)

01862526 SUPPLIER NUMBER: 55941777 (USE FORMAT 7 OR 9 FOR FULL TEXT)
Lichen sclerosus.(Seminar)(Statistical Data Included)
Powell, J J; Wojnarowska, F
The Lancet, 353, 9166, 1777
May 22,
1999
DOCUMENT TYPE: Statistical Data Included PUBLICATION FORMAT: Magazine/Journal ISSN: 0099-5355 LANGUAGE: English RECORD TYPE: Fulltext; Abstract TARGET AUDIENCE: Professional
WORD COUNT: 6271 LINE COUNT: 00515

ABSTRACT: The authors discuss the epidemiology of lichen sclerosis, internationally accepted term for an inflammatory skin disease which is also known as leucoplakia or lichen albus. It is a long-lasting, localized inflammation which usually affects the anogenital area, and risk factors for contracting it are not known. Treatment is difficult.

3/3,AB/11 (Item 11 from file: 149)
DIALOG(R)File 149:TGG Health&Wellness DB(SM)

01802348 SUPPLIER NUMBER: 21245955 (USE FORMAT 7 OR 9 FOR FULL TEXT)
Plasma levels of the soluble fraction of tumor necrosis factor receptor 2 and insulin resistance.
Fernandez-Real, Jose-Manuel; Broch, Montserrat; Ricart, Wifredo; Casamitjana, Roser; Gutierrez, Cristina; Vendrell, Joan; Richart, Cristobal
Diabetes, v47, n11, p1757(6)

Nov,
1998
PUBLICATION FORMAT: Magazine/Journal; Refereed ISSN: 0012-1797
LANGUAGE: English RECORD TYPE: Fulltext TARGET AUDIENCE: Professional
WORD COUNT: 5221 LINE COUNT: 00473

3/3,AB/12 (Item 12 from file: 149)
DIALOG(R)File 149:TGG Health&Wellness DB(SM)

01765815 SUPPLIER NUMBER: 19731336 (USE FORMAT 7 OR 9 FOR FULL TEXT)
Osteoarthritis. (review article)
Creamer, Paul; Hochberg, Marc C.
The Lancet, v350, n9076, p503(7)
August 16,
1997
PUBLICATION FORMAT: Magazine/Journal; Refereed ISSN: 0099-5355
LANGUAGE: English RECORD TYPE: Fulltext; Abstract TARGET AUDIENCE: Professional
WORD COUNT: 6018 LINE COUNT: 00521

ABSTRACT: Osteoarthritis is a common form of arthritis characterized by a softening and loss of joint cartilage and possibly bone. It causes joint pain, tenderness, limitation of movement and may also have an inflammatory component. The risk increases with age and it usually affects the hands, knees, feet and hips. Obesity, occupation and sports participation are also risk factors. Pain can be treated with acetaminophen or non-steroidal anti-inflammatory drugs. Topical creams and corticosteroid joint injections may also be helpful. Physical therapy and joint replacement are other options.

3/3,AB/13 (Item 13 from file: 149)
DIALOG(R)File 149:TGG Health&Wellness DB(SM)

01750179 SUPPLIER NUMBER: 20318160 (USE FORMAT 7 OR 9 FOR FULL TEXT)
Age-related response to interferon alfa treatment in women vs men with chronic hepatitis C virus infection.
Hayashi, Jun; Kishihara, Yasuhiro; Ueno, Kumiko; Yamaji, Kouzaburo; Kawakami, Yasunobu; Furusyo, Norihiro; Sawayama, Yasunori; Kashiwagi, Seizaburo
Archives of Internal Medicine, v158, n2, p177(5)
Jan 26,
1998
PUBLICATION FORMAT: Magazine/Journal; Refereed ISSN: 0003-9926
LANGUAGE: English RECORD TYPE: Fulltext; Abstract TARGET AUDIENCE: Professional
WORD COUNT: 4008 LINE COUNT: 00381

AUTHOR ABSTRACT: Background: Interferon alfa is used widely for patients with chronic hepatitis C virus (HCV) infection. Little is known, however, of the relationship between patients' sex and the effectiveness of interferon alfa treatment in these patients. Methods: We treated 311 patients (199 men and 112 women) with human lymphoblastoid interferon (6 million units subcutaneously every day for 2 weeks and 3 times a week for 22 weeks) and observed them for an additional 6 months. Serum HCV RNA

levels and genotype were tested by polymerase chain reaction before treatment. A liver biopsy was also done. For the purposes of this study, a complete response was defined as the elimination of HCV RNA for at least 6 months after the termination of treatment. Results: The rate of complete response was 27.1% for men and 24.1% for women. With multiple logistic regression analysis, the HCV RNA level (P (is less than) .001), genotype (P (is less than) .001), patients' sex (P (is less than) .05), and the interaction between sex and age were associated with a complete response to interferon alfa. The rate of complete response was 33.3% in men aged 39 years and younger, 25.0% in men aged 40 years and older, 75.0% in women aged 39 years and younger, and 15.6% in women aged 40 years and older. The odds ratio by group was 1.00, 0.72, 4.38, and 0.21, respectively. Conclusions: Our finding that women aged 39 years and younger are responsive to interferon alfa treatment suggests that hormonal activity, in particular the level of estrogen, may be associated with the sustained elimination of HCV. Arch Intern Med. 1998;158:177-181

3/3,AB/14 (Item 14 from file: 149)
DIALOG(R)File 149:TGG Health&Wellness DB(SM)

01717128 SUPPLIER NUMBER: 19748119 (USE FORMAT 7 OR 9 FOR FULL TEXT)
Long-term effects of bisphosphonates on the growing skeleton: studies of young patients with severe osteoporosis.
Brumsen, Caro; Hamdy, Neveen A. T.; Papapoulos, Socrates E.
Medicine, v76, n4, p266(18)
July,
1997
PUBLICATION FORMAT: Magazine/Journal; Refereed ISSN: 0025-7974
LANGUAGE: English RECORD TYPE: Fulltext TARGET AUDIENCE: Professional
WORD COUNT: 9060 LINE COUNT: 00774

3/3,AB/15 (Item 15 from file: 149)
DIALOG(R)File 149:TGG Health&Wellness DB(SM)

01669298 SUPPLIER NUMBER: 18710785 (USE FORMAT 7 OR 9 FOR FULL TEXT)
Cellular changes involved in conversion of normal to malignant breast: importance of the stromal reaction.
Ronnov-Jessen, Lone; Petersen, Ole W.; Bissell, Mina J.
Physiological Reviews, v76, n1, p69(57)
Jan,
1996
PUBLICATION FORMAT: Magazine/Journal ISSN: 0031-9333 LANGUAGE: English
RECORD TYPE: Fulltext; Abstract TARGET AUDIENCE: Professional
WORD COUNT: 48681 LINE COUNT: 03996

ABSTRACT: Breast cancer is characterized by a disorganization of the breast cells and a proliferation of stromal tissue throughout the breast. In the normal breast, a duct is surrounded by luminal epithelial cells that form into myoepithelial cells, which are then surrounded by a basal membrane. Studies of malignant breasts have found evidence that the stem cell for cancer may come from the myoepithelial cells, so that the duct is eventually blocked. Three types of carcinomas have been identified.

3/3,AB/16 (Item 16 from file: 149)
DIALOG(R)File 149:TGG Health&Wellness DB(SM)

01660977 SUPPLIER NUMBER: 18999122 (USE FORMAT 7 OR 9 FOR FULL TEXT)
*Polymorphism"** of the beta(sub 3)-adrenergic receptor gene affects basal metabolic rate in obese Finns.
Sipilainen, Raisa; Uusitupa, Matti; Heikkinen, Sami; Rissanen, Aila; Laakso, Markku
Diabetes, v46, n1, p77(4)
Jan,
1997
PUBLICATION FORMAT: Magazine/Journal ISSN: 0012-1797 LANGUAGE: English
RECORD TYPE: Fulltext TARGET AUDIENCE: Professional
WORD COUNT: 3204 LINE COUNT: 00266

3/3,AB/17 (Item 17 from file: 149)
DIALOG(R)File 149:TGG Health&Wellness DB(SM)

01659586 SUPPLIER NUMBER: 18943719 (USE FORMAT 7 OR 9 FOR FULL TEXT)
High frequency of BRCA1 185delAG *mutation"** in ovarian cancer in Israel.
Modan, Baruch; Gak, Eva; Sade-Bruchim, Revital Bar; Hirsh-Yechezkel, Galit; Theodor, Livia; Lubin, Flora; Ben-Baruch, Gilad; Beller, Uzi; Fishman, Amiram; Dgani, Ram; Menczer, Joseph; Papa, Moshe; Friedman, Eitan
JAMA, The Journal of the American Medical Association, v276, n22, p1823(3)
Dec 11,
1996
PUBLICATION FORMAT: Magazine/Journal ISSN: 0098-7484 LANGUAGE: English
RECORD TYPE: Fulltext; Abstract TARGET AUDIENCE: Professional
WORD COUNT: 2537 LINE COUNT: 00221

ABSTRACT: The presence of the 185delAG *mutation"** in the BRCA1 gene may increase a woman's risk of developing ovarian cancer. The BRCA1 gene has been linked to breast and ovarian cancer. Researchers tested for the gene *mutation"** in 79 women with ovarian cancer, 62 women hospitalized for other reasons and 120 healthy volunteers. Almost 40% of the ovarian cancer patients with a family history had the *mutation"**. Thirteen percent of the ovarian cancer patients with no family history also had the *mutation"**. Only one person among the other two groups had the *mutation"**.
AUTHOR ABSTRACT: Objective.--To determine the role of BRCA1 185deIAG *mutation"** in ovarian carcinogenesis. Design.--Genetic testing of a subset of cases from an ongoing study of ovarian cancer and of controls. Setting.--A community-based case-control incidence study. Subjects.--Seventy-nine patients with ovarian cancer, 62 hospitalized women without cancer (controls), and 120 healthy women participating in a fragile X screening program (also controls), examined for the presence of germline BRCA 1 185deIAG *mutation"**. Main Outcome Measures.--Polymerase chain reaction-amplified BRCA1 exon 2 fragments generated from patients' and controls' blood samples, analyzed by heteroduplex gel shift assay and direct sequence analyses. Results.--The 185deIAG *mutation"** was detected in 38.9% (7/18) of ovarian cancer patients with familial history, and 13.1% (8/61) of family history-negative ovarian cancer cases. Only 1 carder was detected among the 120 healthy controls, and none in the hospital controls. A significant difference in *mutation"** carrier rates between family history-negative cases and control groups of 120 and 62 subjects was

identified (Fisher exact test, P=.001 and P=-.003, respectively). The median age ((+or -)SE) at disease diagnosis was lower among both familial and family historynegative *mutation"** carriers, as compared with *mutation"**-negative, family historynegative cases--50 ((+or -)1.4) vs 60.5 ((+or -)3.5) years old, respectively (hazard ratio, 1.68; 95% confidence interval, 0.94-3.01). Conclusions.-.- Our data are preliminary but suggest that BRCA 1 185deIAG germline *mutation"** is frequent in Israeli ovarian cancer patients, irrespective of family history, and may confer an early-onset phenotype of ovarian cancer. JAMA. 1996;276:1823-1825

3/3,AB/18 (Item 18 from file: 149)
DIALOG(R)File 149:TGG Health&Wellness DB(SM)

01620959 SUPPLIER NUMBER: 18371784 (USE FORMAT 7 OR 9 FOR FULL TEXT)
Transcriptional control of osteoblast growth and differentiation.
Stein, Gary S.; Lian, Jane B.; Stein, Janet L.; Van Wijnen, Andre J.; Montecino, Martin
Physiological Reviews, v76, n2, p593(37)
April,
1996
PUBLICATION FORMAT: Magazine/Journal ISSN: 0031-9333 LANGUAGE: English
RECORD TYPE: Fulltext; Abstract TARGET AUDIENCE: Professional
WORD COUNT: 33889 LINE COUNT: 02799

ABSTRACT: Osteoblasts undergo sequential stages of proliferation and mineralization in the process of development into bone. The proliferation stage is regulated by genes involve in initiating activation of mitosis, including c-fos, c-myc, c-jun, histones and cyclins. The development of the extracellular bone matrix is governed by genes encoding fibronectin, TGF-beta and type I collagen. The last stage, hydroxyapatite and collagenase deposition, is regulated by osteopontin and osteocalcin.

3/3,AB/19 (Item 19 from file: 149)
DIALOG(R)File 149:TGG Health&Wellness DB(SM)

01610139 SUPPLIER NUMBER: 18003876 (USE FORMAT 7 OR 9 FOR FULL TEXT)
Florence Nightingale's fever. (Crimean fever)
Young, D.A.B.
British Medical Journal, v311, n7021, p1697(4)
Dec 23,
1995
PUBLICATION FORMAT: Magazine/Journal ISSN: 0959-8146 LANGUAGE: English
RECORD TYPE: Fulltext; Abstract TARGET AUDIENCE: Professional
WORD COUNT: 3259 LINE COUNT: 00271

ABSTRACT: Founder of modern nursing Florence Nightingale may have suffered from a bacterial infection she contracted during the Crimean War, which would explain her 25 years of convalescence. When no organic disease could be found, she was diagnosed with neurasthenia, which is associated with psychosomatic illness. Her symptoms included insomnia, anorexia, anemia, nervousness, and depression. Her doctors had recommended bed rest, and the nurse followed their advice. Nightingale may have been infected by brucella melitensis, which causes the disease brucellosis. Neurasthenia has been

incorrectly diagnosed in cases of brucellosis. Nightingale's reputation has been tarnished by biographers who wrote that she was a malingerer who used her sickness for publicity.

3/3,AB/20 (Item 20 from file: 149)
DIALOG(R)File 149:TGG Health&Wellness DB(SM)

01610114 SUPPLIER NUMBER: 17931220 (USE FORMAT 7 OR 9 FOR FULL TEXT)
Osteoarthritis: a continuing challenge.
Sack, Kenneth E.
The Western Journal of Medicine, v163, n6, p579(8)
Dec,
1995
PUBLICATION FORMAT: Magazine/Journal ISSN: 0093-0415 LANGUAGE: English
RECORD TYPE: Fulltext; Abstract TARGET AUDIENCE: Professional
WORD COUNT: 7372 LINE COUNT: 00661

AUTHOR ABSTRACT: Osteoarthritis is a disorder of cartilage that affects almost 85% of the population by age 75. A lack of rigorous clinical and radiographic criteria for defining the disorder makes precise determination of its prevalence impossible. The process of wear and tear explains many manifestations of osteoarthritis, but it does not account for some of the clinical findings or the biochemical changes in osteoarthritic cartilage. Thus, other factors such as heredity, hormones, and diet may play a role' Treatment consists of teaching patients about their disease, alleviating pain, and preserving joint function. Nonsteroidal anti-inflammatory drugs may be no more effective than simple analgesics in relieving the pain of this disorder. Moreover, some nonsteroidal anti-inflammatory drugs can adversely affect cartilage metabolism, and most are possibly dangerous in elderly patients. Drugs that inhibit the production or activity of chondrolytic enzymes can slow the degeneration of cartilage in some animals, but their effects on humans with osteoarthritis are unproved. The surgical repair of severely damaged joints can have gratifying results.

3/3,AB/21 (Item 21 from file: 149)
DIALOG(R)File 149:TGG Health&Wellness DB(SM)

01607615 SUPPLIER NUMBER: 17798844 (USE FORMAT 7 OR 9 FOR FULL TEXT)
Genetic variation, nutrition, and chronic diseases. (Genetic Variation and Nutrition, part 2)
Simopoulos, Artemis P.
Nutrition Today, v30, n5, p194(13)
Sept-Oct,
1995
PUBLICATION FORMAT: Magazine/Journal ISSN: 0029-666X LANGUAGE: English
RECORD TYPE: Fulltext; Abstract TARGET AUDIENCE: Consumer
WORD COUNT: 8286 LINE COUNT: 00707

AUTHOR ABSTRACT: This review of current knowledge of the role of genetic variation and nutrition in the prevention and treatment of chronic diseases builds on Part 1, which appeared in the August issue of Nutrition Today. General dietary recommendations are appropriate in countries where the basic problem is one of undernutrition. However, in Western societies, where overnutrition and sedentary lifestyles predominate, universal dietary

recommendations for the prevention of chronic diseases are inappropriate. Rather, specific dietary recommendations targeted to the individual at risk based on family history and genetic predisposition are in order for both prevention and treatment of chronic diseases.

3/3,AB/22 (Item 22 from file: 149)
DIALOG(R)File 149:TGG Health&Wellness DB(SM)

01605209 SUPPLIER NUMBER: 17623519 (USE FORMAT 7 OR 9 FOR FULL TEXT)
Breast cancer: cause and prevention.(Review Article)
Hulka, Barbara S.; Stark, Azadeh T.
The Lancet, v346, n8979, p883(5)
Sept 30,
1995
PUBLICATION FORMAT: Magazine/Journal ISSN: 0099-5355 LANGUAGE: English
RECORD TYPE: Fulltext; Abstract TARGET AUDIENCE: Professional
WORD COUNT: 5067 LINE COUNT: 00416

ABSTRACT: The prevention of breast cancer is challenging current research efforts. Breast cancer is the most common cancer among women of the industrialized world. Some of the known causes include a family history of breast cancer, genetic and hormonal factors, dietary and nutritional aspects, lifestyle and environmental risks. Primary prevention aims to reduce identified breast cancer risks, such as limiting oral contraceptives and hormone replacement therapy. Tamoxifen, an anti-estrogen drug, seems to prevent cancer spread in some women and is currently undergoing testing. Pregnancy early in life may protect against breast cancer but fails to be a practical solution. Some lifestyle options are more feasible for the individual and include weight control, exercise, alcohol abstinence, vitamin A supplementation, and dietary fat reduction. Long-term nursing may not be an option for women in developed countries.

3/3,AB/23 (Item 23 from file: 149)
DIALOG(R)File 149:TGG Health&Wellness DB(SM)

01364546 SUPPLIER NUMBER: 12448822 (USE FORMAT 7 OR 9 FOR FULL TEXT)
Viseral obesity in men: associations with glucose tolerance, plasma insulin, and lipoprotein levels.
Pouliot, Marie-Christine; Despres, Jean-Pierre; Nadeau, Andre; Moorjani, Sital; Prud'Homme, Denis; Lupien, Paul J.; Tremblay, Angelo; Bouchard, Claude
Diabetes, v41, n7, p826(9)
July,
1992
PUBLICATION FORMAT: Magazine/Journal ISSN: 0012-1797 LANGUAGE: English
RECORD TYPE: Fulltext TARGET AUDIENCE: Professional
WORD COUNT: 5067 LINE COUNT: 00528

3/3,AB/24 (Item 24 from file: 149)
DIALOG(R)File 149:TGG Health&Wellness DB(SM)

01293058 SUPPLIER NUMBER: 10346593 (USE FORMAT 7 OR 9 FOR FULL TEXT)

Overview of osteoporosis.
Riggs, B. Lawrence
The Western Journal of Medicine, v154, n1, p63(15)
Jan,
1991
PUBLICATION FORMAT: Magazine/Journal ISSN: 0093-0415 LANGUAGE: English
RECORD TYPE: Fulltext; Abstract TARGET AUDIENCE: Professional
WORD COUNT: 10468 LINE COUNT: 01091

ABSTRACT: Osteoporosis occurs when there is decrease in the bone mass to a level where the bone can no longer the body. About 1.5 million fractures caused by osteoporosis occur in the United States each year, usually involving the hip, spine, or forearm. One in three women over 65 will suffer fractures of the spine, and 15 percent of white women, who are at higher risk, will suffer a hip fracture. The costs of these injuries is several billion dollars per year, and hip fractures are fatal in 12 to 20 percent of the cases. Fifty percent of the survivors never walk unaided again, and one in four must remain in a nursing home. This article reviews the process of bone formation and subsequent age-related loss, and the causes of osteoporosis. Osteoporosis in children and young adults is mentioned. A distinction is made between postmenopausal (type I) osteoporosis and osteoporosis caused by other factors, such as endocrine disorders (e.g., hyperthyroidism), gastrointestinal diseases (e.g., malabsorption diseases or anorexia), bone marrow disorders, connective tissue diseases, and others. The symptoms are described and the diagnostic procedure is outlined, including X-ray images. Drug therapy with estrogen is described, as well as calcium, vitamin D and calcitonin supplementation, and the use of biphosphonates, anabolic steroids, and sodium fluoride. Diet and exercise are mentioned but not stressed. Prevention is the only effective approach, and calcium intake should be at least 1,000 mg per day, perhaps more. Cigarettes and alcohol, both bone toxins, should be eliminated. The authors state that estrogen replacement beginning at *menopause** is the most effective preventive, although this is highly controversial because of the increase in breast cancer and other ill effects. (Consumer Summary produced by Reliance Medical Information, Inc.)

3/3,AB/25 (Item 1 from file: 144)
DIALOG(R)File 144:Pascal

15354424 PASCAL No.: 02-0041823
*Polymorphisms** for *interleukin**-*1** beta (*IL**-*1** beta)-511 promoter, *IL**-*1** beta exon 5, and *IL**-*1** receptor antagonist : Nonassociation with endometriosis
HSIEH Yao-Yuan; CHANG Chi-Chen; TSAI Fuu-Jen; WU Jer-Yuarn; SHI Yi-Ru; TSAI Horng-Der; TSAI Chang-Hai
Department of Obstetrics and Gynecology, China Medical College Hospital, Taichung, Taiwan; Department of Pediatrics and Medical Genetics, China Medical College Hospital, Taichung, Taiwan
Journal: Journal of assisted reproduction and genetics, 2001, 18 (9) 506-511
Language: English
Purpose: We aimed to investigate if *interleukin**-*1** beta (*IL**-*1** beta) and *IL**-*1** receptor antagonist (IL-1Ra) gene *polymorphism** could be used as markers of susceptibility in endometriosis. Materials and Methods: Women were divided into two groups: 1) endometriosis (n = 120); 2) nonendometriosis groups (n = 103).

*Polymorphisms"** for *IL"**-*1"** beta -511 promoter, *IL"**-*1"** beta exon 5, and IL-1Ra were detected by polymerase chain reaction. Genotypes and allelic frequencies for these *polymorphisms"** in both groups were compared. Results: Proportions of different IL- I and IL- 1Ra *polymorphisms"** in both groups were nonsignificantly different. Proportions of C homozygote/heterozygote/T homozygote for *IL"**-*1"** beta -511 promoter in both groups were 1) 21.6/59.1/19.1% and 2) 26.2/50.5/23.3%. Proportions of E1 homozygote/ heterozygote/E2 homozygote for *IL"**-*1"** beta exon 5 in both groups were 1) 91.6/5/3.3% and 2) 95.15/4.85/0%. Allele I/ II/IV/V for IL-1Ra in both groups were 1) 92.5/5.4/1.6/0.4% and 2) 95.1/3.9/1/0%. Conclusions: Association of endometriosis with *IL"**-*1"** beta 511 promoter, *IL"**-*1"** beta exon 5, and *IL"**-*1"** receptor antagonist gene *polymorphisms"** doesn't exist. These *polymorphisms"** are not useful markers for prediction of endometriosis susceptibility.

3/3,AB/26 (Item 2 from file: 144)
DIALOG(R)File 144:Pascal

13768522 PASCAL No.: 98-0481155

*Allelic"** *variation"** at the *interleukin"**-*1"** receptor antagonist gene is associated with early postmenopausal bone loss at the spine

KEEN R W; WOODFORD-RICHENS K L; LANCHBURY J S; SPECTOR T D
Twin & Genetic Epidemiology Research Unit, St. Thomas' Hospital, London, United Kingdom; Molecular Immunogenetics Unit, Thomas Guy House, UMDS, Guy's Hospital, London, United Kingdom

Journal: Bone : (New York), 1998, 23 (4) 367-371
Language: English

Genetic factors play an important role in determining bone mineral density (BMD) in later life, with the genetic influence mediated through effects on both peak mass and on age- and *menopause"**-related bone loss. At *menopause"** there is an increase in the production and activity of various cytokines and growth factors within the bone microenvironment. The activity of *interleukin"**-*1"** (IL-I), a powerful stimulant of osteoclastic bone resorption, is increased in estrogen-deficient states with increased production of *IL"**-*1"** and inhibition of the *IL"**-*1"** receptor antagonist (IL-1ra). Treatment with IL-1ra blocks the hone loss associated with ovariectomy in animals and the *IL"**-*1"** receptor antagonist gene (*IL"**-*1RN"**) is therefore a potential candidate gene for the regulation of postmenopausal bone loss. We examined the relationship between annual rates of change in BMD over 5 years and an 86 bp variable number tandem-repeat *polymorphism"** of the *IL"**-*1RN"** gene in 108 early postmenopausal women. All women were within 5 years of a natural *menopause"** at the study's onset, healthy, and not on hormone replacement therapy or other medication known to affect bone metabolism. BMD was measured annually over the 5 year study period at the lumbar spine and femoral neck using dual-energy X-ray absorptiometry. Three alleles were identified (A1 = 4 repeats, A2 = 2 repeats, A3 = 5 repeats), with five genotypes observed: A1A1 (41.7%), A1A2 (45.4%), A2A2 (6.5%), A1A3 (2.8%), and A2A3 (3.7%). For analysis, alleles were collapsed into a biallelic system grouping the A1 and A3 alleles. There was no significant relationship between the *IL"**-*IRN"** genotypes and baseline bone mass at either the spine or hip. *IL"**-*1RN"** genotype was significantly

associated with annual rates of change in spinal bone mass ($p < 0.05$), and this finding remained significant after adjustment for age, weight, and baseline BMD. Carriage of at least one copy of the A2 allele was associated with reduced bone loss at the spine (mean change in BMD +- SD: -0.81 +- 1.46%/year) when compared with noncarriage of the A2 allele (mean change -1.38 +- 1.48%/year), $p = 0.05$. We therefore conclude that *allelic"** *variation"** at the *IL"**-*1RN"** locus is associated with differential rates of early postmenopausal bone loss at the spine. Further research will be required to clarify the mechanisms underlying these findings and to determine whether this association translates into a significant long-term effect on BMD and fracture in later life.

3/3,AB/27 (Item 1 from file: 76)
DIALOG(R)File 76:Life Sciences Collection

02658351 5184984
Linkage of human tumor necrosis factor-alpha to human osteoporosis by sib-pair analysis
Ota, N.; Hunt, S.C.; Nakajima, T.; Suzuki, T.; Hosoi, T.; Orimo, H.; Shirai, Y.; Emi, M.
Department of Molecular Biology, Institute of Gerontology, Nippon Medical School, 1-396 Kosugi-cho, Nakahara-ku, Kawasaki 211-8533, Japan
Genes and Immunity vol. 1, no. 4, pp. 260-264 (2000)
ISSN: 1466-4879
DOCUMENT TYPE: Journal article LANGUAGE: ENGLISH
SUBFILE: Genetics Abstracts; Immunology Abstracts

Osteoporosis as well as osteopenia are common human conditions considered to result from the interplay of multiple genetic and environmental factors. Twin and family studies have yielded strong correlation between measures of bone mass and a number of genetic factors. Certain genes (eg, cytokines such as *interleukin"**-*1"**, interleukin-6, or tumor necrosis factor-alpha) are capable of regulating metabolism, formation, and resorption of bone all processes that determine bone mass. We tested 192 sib-pairs of adult Japanese women from 136 families for genetic linkage between osteoporosis and osteopenia phenotypes and *allelic"** *variants"** at the tumor necrosis factor-alpha (TNFA) locus, using a dinucleotide-repeat *polymorphism"** located near the gene. The TNFA locus showed evidence for linkage to osteoporosis, with mean allele sharing of 0.478 ($P = 0.30$) in discordant pairs and 0.637 ($P = 0.001$) in concordant affected pairs. Linkage with osteopenia was also significant in concordant affected pairs ($P = 0.017$). Analyses limited to the post-*menopausal"** women in our cohort showed similar or even stronger linkage for both phenotypes. The results provide evidence that genetic variations within the TNFA locus or adjacent genes affect regulation of mineral metabolism in bone and some of them confer risk for osteoporosis in adult women.

3/3,AB/28 (Item 2 from file: 76)
DIALOG(R)File 76:Life Sciences Collection

02533046 4749889

TGF- beta 1 stimulates expression of the aromatase (CYP19) gene in human osteoblast-like cells and THP-1 cells

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Molecular and Cellular Endocrinology vol. 160, no. 1-2, pp. 123-133 (2000)
ISSN: 0303-7207
DOCUMENT TYPE: Journal article LANGUAGE: ENGLISH
SUBFILE: Genetics Abstracts; Calcium & Calcified Tissue Abstracts

Recent evidence has shown that bone is not only a target of estrogen action but also a source of local estrogen production. Bone cells such as osteoblasts express aromatase (P450arom) and the expression of P450arom in osteoblasts is positively regulated in a tissue specific fashion, as in the case of other tissues which express P450arom. To clarify the physiological factors regulating expression of P450arom in bone, we tested TGF- beta 1 using osteoblast-like cells obtained from human fetuses as well as THP-1 cells. TGF- beta 1 increased *IL"**-*I"** beta +DEX- induced aromatase activity in osteoblast-like cells, while it inhibited activity in skin fibroblasts. Similar enhancement of aromatase activity by TGF- beta 1 was found in DEX-stimulated THP-1 cells and this cell line was used for further experiments. In THP-1 cells, TGF- beta 1 enhanced DEX-induced aromatase activity almost linearly by 12 h and thereafter. Increased levels of P450arom transcripts were also demonstrated by RT-PCR at 3 h of TGF- beta 1 treatment and thereafter. Cyclohexamide abolished enhancement of activity but did not inhibit the accumulation of P450arom transcripts induced by TGF- beta 1. Increase in P450arom expression by TGF- beta 1 was attributable to expression driven by promoter I.4. TGF- beta 1 did not change the half life of P450arom transcripts. To identify the cis-acting elements responsible for TGF- beta 1 action on aromatase expression, transient transfection assays were performed using a series of deletion constructs for promoter I.4 (P450-I.4/Luc). Two constructs (-410/+14 and-340/+14) that contain a functional glucocorticoid response element (GRE) and downstream sequence showed significant increase of luciferase activity in response to TGF- beta 1. Deletion and *mutation"** of the GRE in P450-I.4/Luc (-340/+14) abolished the TGF- beta 1. The luciferase activity of a (GRE) sub(1)-SV40/Luc construct was also stimulated by TGF-beta 1. These results indicate that TGF- beta 1 increases the expression of P450arom at the level of transcription through promoter I.4, at least in part via an enhancement of transactivation activity of the GR in THP-1 cells. TGF- beta 1 is suggested to be one of the physiological up-regulatory factors of bone aromatase.

00110821

Phacoemulsification in Difficult and Challenging Cases - Foreign Body Entrapment in Radial Keratotomy Incisions (ARTICLE)

Archives of Ophthalmology
June, 1999; Case Reports and Small Case: tzh836
LINE COUNT: 00474

3/3,AB/30 (Item 2 from file: 442)
DIALOG(R)File 442:AMA Journals

00105596

Autoimmune Endocrine Disease (ARTICLE)

BAKER, JAMES R.
JAMA, The Journal of the American Medical Association
December 10, 1997; 22: tzj1931
LINE COUNT: 00706

Autoimmune endocrine diseases are serious disorders that utilize immense health care resources and cause tremendous disability. They include type 1 diabetes mellitus, thyroiditis, Graves disease, Addison disease, and polyglandular syndromes. Analysis of the basis of autoimmune diseases has been aided by the application of new knowledge in immunologic physiology. Recent investigations using these techniques have revealed complicated disorders that have varied pathogenesis and complex genetic predispositions. While the mainstay of treatment for these diverse diseases remains the replacement of hormones produced by the damaged endocrine organ, investigations into the pathogenesis of these disorders provide hope for the development of specific therapeutic measures to block their pathologic basis.JAMA. 1997;278:1931-1937

3/3,AB/31 (Item 3 from file: 442)
DIALOG(R)File 442:AMA Journals

00085620

The Study of Gene-Environment Interactions That Influence Thrombosis and Fibrinolysis Genetic Variation at the Loci for Factor VII and Plasminogen Activator Inhibitor-1 (ARTICLE)

HUMPHRIES, STEVE E.; LANE, ANNE; DAWSON, SALLY; GREEN, FIONA R.
Archives of Pathology and Laboratory Medicine
December, 1992 Thrombosis--Humphries et al; p1322
LINE COUNT: 00679

We describe studies on variation in the genes coding for factor VII (FVII) and plasminogen activator inhibitor-1 (PAI-1) that influence levels of these proteins in the blood. For FVII, we have identified agenetic *polymorphism"** that results in the substitution of arginine at residue 353 to glutamine. The frequency of the glutamine allele is approximately 0.1 in samples of individuals from the United Kingdom (n=777) and the United States (n=140) and in Afro-Caribbeans (n=49), and is significantly higher in a sample of individuals from the Indian subcontinent (n=53). In all samples, carriers of the glutamineallele had levels of FVII coagulant activity 20% to 25% lower than those with only the arginine allele. These differences were highly statistically significant in the United Kingdom sample. This effect was consistent in healthy men and women and in those

with coronary artery disease. In individuals homozygous for the glutamine allele, both FVII coagulant activity and antigen are low, and the mechanism of the association appears likely to be due to an effect on secretionfrom the liver or stability in the plasma. In individuals in the general population FVII coagulant activity is positively correlated with levels of plasma triglycerides, due to the effect of such lipoproteins on activation of FVII. This relationship appears weaker in individuals carrying the glutamine allele, and since elevated FVIIcoagulant antibody is associated with risk of thrombosis, this is an example of how environmental factors may interact with an individual's genotype to determine his or her thrombotic risk. Roughly 20% of the general population are carriers of the glutamine allele and are likely to be genetically protected from such risk. ForPAI-1, we have recently shown that variation at the PAI gene locus, detected as DNA *polymorphisms"**, is associated with between-individual differences in levels of PAI-1. We have now detected a common sequence change in the promoter region of the gene that explains partof this effect. The sequence change is at position 675, where a fifthguanine (5G allele) has been inserted into a run of four guanines (4Gallele) when compared with published sequences. In a sample of both 83 healthy individuals and 105 young patients with coronary artery disease from Sweden, the frequency of the 4G allele is roughly 0.5, and those individuals homozygous for the 4G allele have higher levelsof PAI-1 than those with other genotypes (29% higher). Preliminary data show that the 5G allele binds a hepatic nuclear protein that the4G allele does not, suggesting that the mechanism of the effect may be due to a direct effect of the sequence change on transcription of the PAI-1 gene. Expression of the PAI-1 gene is known to be modulatedby cytokines produced in the acute-phase response, and statistical analysis shows evidence for a strongly positive relationship between PAI-1 levels and markers of the acute phase in individuals with the genotype 4G/4G (correlation with fibrinogen, 0.33 in patients, 0.42 in healthy group) and a weaker or negative relationship in those withother genotypes (for the 5G/5G group, -0.35, and -0.18, respectively). These differences were statistically significiant in the healthy group, and represent a second situation where interactionbetween environmental factors and genetic variation may be important in determining an individual's levels of PAI-1 over time and thus risk of a thrombotic event due to reduced fibrinolytic potential. (Arch Pathol Lab Med. 1992;116:)

3/3,AB/32 (Item 4 from file: 442)
DIALOG(R)File 442:AMA Journals

00085617

Thrombosis and Cardiovascular Risk in the Elderly (ARTICLE)

TRACY, RUSSELL P.
Archives of Pathology and Laboratory Medicine
December, 1992; Original Article: p1307
LINE COUNT: 00557

Current concepts of cardiovascular disease pathophysiology include a prominent role for thrombosis as a key factor. Thrombosis is not onlythe usual precipitant to a clinical event, but it may also be involved in atherosclerotic plaque development throughout most of theadult years. However, our understanding of thrombotic risk factors, especially in the

elderly, is poor and research has just begun in this area. Fibrinogen has been clearly established as an independent risk factor in the middle aged, but there are conflicting data concerning older persons. Factor VII and plasminogen activator inhibitor-1 look promising as risk factors in the middle aged, but there are no data currently available concerning the status of these factors in the elderly. Many associations exist between the thrombotic risk factors and other cardiovascular risk factors such asplasma lipids and glucose intolerance, making the establishment of independence difficult, and little is known about how these differentfactors may interact in older individuals. Ongoing studies should provide many answers in the near future. (Arch Pathol Lab Med. 1992;116:)

3/3,AB/33 (Item 5 from file: 442)
DIALOG(R)File 442:AMA Journals

00025544

Glucan-Induced Keratoderma in Acquired Immunodeficiency Syndrome (STUDIES)

DUVIC, MADELEINE; REISMAN, MARK; FINLEY, VICTORIA; RAPINI, RONALD; DILUZIO, NICHOLAS R.; MANSELL, PETER W. A.
Archives of Dermatology
June, 1987; 123: 751-7561987;
LINE COUNT: 00204 WORD COUNT: 02828

ABSTRACT: Six of 20 patients with acquired immunodeficiency syndrome (AIDS) or AIDS-related complex receiving intravenous infusions of soluble glucan (beta-1-3 polyglucose) developed a keratoderma of the palms and soles. The eruption began during the first two weeks of therapy and resolved two to four weeks after its discontinuation. The eruption was different in appearance from our previously reported keratoderma blennorrhagica in AIDS-associated psoriasis. None of the other 735 patients with AIDS or AIDS-related complex not treated with soluble glucan developed a similar keratoderma. The correlation between receiving glucan and the hyperkeratosis is highly significant. Since glucan is a naturally occurring component of the cell walls of yeast, fungus, and some bacterial organisms, recognition of its ability to induce such a striking reaction pattern may be of general significance and interest, although the reaction itself may be limited to patients with AIDS.

3/3,AB/34 (Item 1 from file: 444)
DIALOG(R)File 444:New England Journal of Med.

00122140

Mechanisms of Disease: Production and Actions of Estrogens (Review Article)

Gruber, Christian J.; Tschugguel, Walter; Schneeberger, Christian; Huber, Johannes C.
The New England Journal of Medicine
Jan 31, 2002; 346 (5),pp 340-352

LINE COUNT: 00466 WORD COUNT: 06443

3/3,AB/35 (Item 2 from file: 444)
DIALOG(R)File 444:New England Journal of Med.

00120379

Mechanisms of Disease: Plasminogen-Activator Inhibitor Type 1 and Coronary Artery Disease (Review Articles)

Kohler, Hans P.; Grant, Peter J.
The New England Journal of Medicine
Jun 15, 2000; 342 (24),pp 1792-1801
LINE COUNT: 00467 WORD COUNT: 06450

3/3,AB/36 (Item 3 from file: 444)
DIALOG(R)File 444:New England Journal of Med.

00120065

Cytokeratin-Positive Cells in the Bone Marrow and Survival of Patients with Stage I, II, or III Breast Cancer (Original Articles)

Braun, Stephan; Pantel, Klaus; Muller, Peter; Janni, Wolfgang; Hepp, Florian; Kentenich, Christina R.M.; Gastroph, Stephan; Wischnik, Artur ; Dimpfl, Thomas; Kindermann, Gunter; Riethmuller, Gert; Schlimok, Gunter.
The New England Journal of Medicine
Feb 24, 2000; 342 (8),pp 525-533
LINE COUNT: 00378 WORD COUNT: 05223

Abstract

Background: Cytokeratins are specific markers of epithelial cancer cells in bone marrow. We assessed the influence of cytokeratin-positive micrometastases in the bone marrow on the prognosis of women with breast cancer.

Methods: We obtained bone marrow aspirates from both upper *iliac"** crests of 552 patients with stage I, II, or III breast cancer who underwent complete resection of the tumor and 191 patients with nonmalignant disease. The specimens were stained with the monoclonal antibody A45-B/B3, which binds to an antigen on cytokeratins. The median follow-up was 38 months (range, 10 to 70). The primary end point was survival.

Results: Cytokeratin-positive cells were detected in the bone marrow specimens of 2 of the 191 control patients with nonmalignant conditions (1 percent) and 199 of the 552 patients with breast cancer (36 percent). The presence of occult metastatic cells in bone marrow was unrelated to the presence or absence of lymph-node metastasis (P=0.13). After four years of follow-up, the presence of micrometastases in bone marrow was associated with the occurrence of clinically overt distant metastasis and death from cancer-related causes (P<0.001), but not with locoregional relapse (P=0.77). Of 199 patients with occult metastatic cells, 49 died of cancer, whereas of 353 patients without such cells, 22 died of cancer-related

causes (P<0.001). Among the 301 women without lymph-node metastases, 14 of the 100 with bone marrow micrometastases died of cancer-related causes, as did 2 of the 201 without bone marrow micrometastases (P<0.001). The presence of occult metastatic cells in bone marrow, as compared with their absence, was an independent prognostic indicator of the risk of death from cancer (relative risk, 4.17; 95 percent confidence interval, 2.51 to 6.94; P<0.001), after adjustment for the use of systemic adjuvant chemotherapy.

Conclusions: The presence of occult cytokeratin-positive metastatic cells in bone marrow increases the risk of relapse in patients with stage I, II, or III breast cancer. (N Engl J Med 2000;342:525-33.)

3/3,AB/37 (Item 4 from file: 444)
DIALOG(R)File 444:New England Journal of Med.

00119835

Metastasizing Aggressive Angiomyxoma (Correspondence)

Siassi, Ramin Michael; Papadopoulos, Thomas; Matzel, Klaus E.
The New England Journal of Medicine
Dec 2, 1999; 341 (23),p 1772
LINE COUNT: 00049 WORD COUNT: 00681

3/3,AB/38 (Item 5 from file: 444)
DIALOG(R)File 444:New England Journal of Med.

00117127

Brief Report: Effect of Testosterone and Estradiol in a Man with Aromatase Deficiency (Original Articles)

Carani, Cesare; Qin, Kenan; Simoni, Manuela; Faustini-Fustini, Marco; Serpente, Stefania; Boyd, Jeff; Korach, Kenneth S.; Simpson, Evan R.
The New England Journal of Medicine
Jul 10, 1997; 337 (2),pp 91-95
LINE COUNT: 00324 WORD COUNT: 04477

3/3,AB/39 (Item 6 from file: 444)
DIALOG(R)File 444:New England Journal of Med.

00116949

Seminars in Medicine of the Beth Israel Deaconess Medical Center: Neuroendocrine Responses to Starvation and Weight Loss (Review Article)

Schwartz, Michael W.; Seeley, Randy J.
The New England Journal of Medicine
Jun 19, 1997; 336 (25),pp 1802-1811
LINE COUNT: 00565 WORD COUNT: 07803

3/3,AB/40 (Item 7 from file: 444)
DIALOG(R)File 444:New England Journal of Med.

00116161

Bone Mineral Density in Women with Depression (Original Articles)

Michelson, David; Stratakis, Constantine; Hill, Lauren; Reynolds, James; Galliven, Elise; Chrousos, George; Gold, Philip.
The New England Journal of Medicine
Oct 17, 1996; 335 (16),pp 1176-1181
LINE COUNT: 00241 WORD COUNT: 03333

Abstract

Background: Depression is associated with alterations in behavior and neuroendocrine systems that are risk factors for decreased bone mineral density. This study was undertaken to determine whether women with past or current major depression have demonstrable decreases in bone density.

Methods: We measured bone mineral density at the hip, spine, and radius in 24 women with past or current major depression and 24 normal women matched for age, body-mass index, *menopausal"** status, and race, using dual-energy x-ray absorptiometry. We also evaluated cortisol and growth hormone secretion, bone metabolism, and vitamin D-receptor alleles.

Results: As compared with the normal women, the mean (+/-SD) bone density in the women with past or current depression was 6.5 percent lower at the spine (1.00+/-0.15 vs. 1.07+/-0.09 g per square centimeter, P = 0.02), 13.6 percent lower at the femoral neck (0.76+/-0.11 vs. 0.88+/-0.11 g per square centimeter, P<0.001), 13.6 percent lower at Ward's triangle (0.70+/-0.14 vs. 0.81+/-0.13 g per square centimeter, P<0.001), and 10.8 percent lower at the trochanter (0.66+/-0.11 vs. 0.74+/-0.08 g per square centimeter, P<0.001). In addition, women with past or current depression had higher urinary cortisol excretion (71+/-29 vs. 51+/-19 microg per day 196+/-80 vs. 141+/-52 nmol per day], P = 0.006), lower serum osteocalcin concentrations (P = 0.04), and lower urinary excretion of deoxypyridinoline (P = 0.02).

Conclusions: Past or current depression in women is associated with decreased bone mineral density. (N Engl J Med 1996;335:1176-81.)

3/3,AB/41 (Item 8 from file: 444)
DIALOG(R)File 444:New England Journal of Med.

00114230

Medical Progress: Recent Advances in Radiation Oncology (Review Article)

Lichter, Allen S.; Lawrence, Theodore S.
The New England Journal of Medicine
Feb 9, 1995; 332 (6),pp 371-379
LINE COUNT: 00498 WORD COUNT: 06882

3/3,AB/42 (Item 9 from file: 444)
DIALOG(R)File 444:New England Journal of Med.

00108615

Hyperhomocysteinemia: An Independent Risk Factor For Vascular Disease (Original Articles)

Clarke, Robert; Daly, Leslie; Robinson, Killian; Naughten, Eileen; Cahalane, Seamus; Fowler, Brian; Graham, Ian.
The New England Journal of Medicine
Apr 25, 1991; 324 (17),pp 1149-1155
LINE COUNT: 00375 WORD COUNT: 05183

Abstract

Background. Hyperhomocysteinemia arising from impaired methionine metabolism, probably usually due to a deficiency of cystathionine beta-synthase, is associated with premature cerebral, peripheral, and possibly coronary vascular disease. Both the strength of this association and its independence of other risk factors for cardiovascular disease are uncertain. We studied the extent to which the association could be explained by heterozygous cystathionine beta-synthase deficiency.

Methods. We first established a diagnostic criterion for hyperhomocysteinemia by comparing peak serum levels of homocysteine after a standard methionine-loading test in 25 obligate heterozygotes with respect to cystathionine beta-synthase deficiency (whose children were known to be homozygous for homocystinuria due to this enzyme defect) with the levels in 27 unrelated age- and sex-matched normal subjects. A level of 24.0 micromol per liter or more was 92 percent sensitive and 100 percent specific in distinguishing the two groups. The peak serum homocysteine levels in these normal subjects were then compared with those in 123 patients whose vascular disease had been diagnosed before they were 55 years of age.

Results. Hyperhomocysteinemia was detected in 16 of 38 patients with cerebrovascular disease (42 percent), 7 of 25 with peripheral vascular disease (28 percent), and 18 of 60 with coronary vascular disease (30 percent), but in none of the 27 normal subjects. After adjustment for the effects of conventional risk factors, the lower 95 percent confidence limit for the odds ratio for vascular disease among the patients with hyperhomocysteinemia, as compared with the normal subjects, was 3.2. The geometric-mean peak serum homocysteine level was 1.33 times higher in the patients with vascular disease than in the normal subjects (P = 0.002). The presence of cystathionine beta-synthase deficiency was confirmed in 18 of 23 patients with vascular disease who had hyperhomocysteinemia.

Conclusions. Hyperhomocysteinemia is an independent risk factor for vascular disease, including coronary disease, and in most instances is probably due to cystathionine beta-synthase deficiency. (N Engl J Med 1991; 324:1149-55.)

3/3,AB/43 (Item 10 from file: 444)
DIALOG(R)File 444:New England Journal of Med.

00104423

Case 13-1988: Pelvic Mass in a 42-Year-Old Woman with a History of Salmonella Sepsis (Case Records of the Massachusetts General Hospital)

Stubblefield, Phillip G.; Bell, Debra A.
The New England Journal of Medicine
March 31, 1988; 318 (13),pp 835-842
LINE COUNT: 00638 WORD COUNT: 08804

3/3,AB/44 (Item 1 from file: 16)
DIALOG(R)File 16:Gale Group PROMT(R)

08927282 Supplier Number: 77416504
PART II Prescription for success.(pharmaceuticals in development)(Illustration)
News, Med Ad
Med Ad News, v20, n7, pNA
July, 2001
Language: English Record Type: Fulltext
Article Type: Illustration
Document Type: Magazine/Journal; Trade
Word Count: 34591

3/3,AB/45 (Item 2 from file: 16)
DIALOG(R)File 16:Gale Group PROMT(R)

08927281 Supplier Number: 77416502
PART I Prescription for success.(developing new pharmaceuticals)(Illustration)
News, Med Ad
Med Ad News, v20, n7, p3
July, 2001
Language: English Record Type: Fulltext
Article Type: Illustration
Document Type: Magazine/Journal; Trade
Word Count: 27870

3/3,AB/46 (Item 1 from file: 98)
DIALOG(R)File 98:General Sci Abs/Full-Text

04755389 H.W. WILSON RECORD NUMBER: BGSA02005389
Aromatase--a brief overview.
Simpson, Evan R
Clyne, Colin; Rubin, Gary
Annual Review of Physiology v. 64 (2002) p. 93-127
SPECIAL FEATURES: bibl il ISSN: 0066-4278
LANGUAGE: English
COUNTRY OF PUBLICATION: United States
WORD COUNT: 16532

ABSTRACT: There is growing awareness that androgens and estrogens have general metabolic roles that are not directly involved in reproductive processes. These include actions on vascular function, lipid and

carbohydrate metabolism, as well as bone mineralization and epiphyseal closure in both sexes. In postmenopausal women, as in men, estrogen is no longer solely an endocrine factor but instead is produced in a number of extragonadal sites and acts locally at these sites in a paracrine and intracrine fashion. These sites include breast, bone, vasculature, and brain. Within these sites, aromatase action can generate high levels of estradiol locally without significantly affecting circulating levels. Circulating C19 steroid precursors are essential substrates for extragonadal estrogen synthesis. The levels of these androgenic precursors decline markedly with advancing age in women, possible from the mid-to-late reproductive years. This may be a fundamental reason why women are at increased risk for bone mineral loss and fracture, and possibly decline of cognitive function, compared with men. Aromatase expression in these various sites is under the control of tissue-specific promotors regulated by different cohorts of transcription factors. Thus in principle, it should be possible to develop selective aromatase modulators (SAMs) that block aromatase expression, for example, in breast, but allow unimpaired estrogen synthesis in other tissues such as bone. Reprinted by permission of the publisher.

3/3,AB/47 (Item 2 from file: 98)
DIALOG(R)File 98:General Sci Abs/Full-Text

04265301 H.W. WILSON RECORD NUMBER: BGSA00015301
Leptin.
Ahima, Rexford S
Flier, Jeffrey S
Annual Review of Physiology v. 62 (2000) p. 413-37
SPECIAL FEATURES: bibl il ISSN: 0066-4278
LANGUAGE: English
COUNTRY OF PUBLICATION: United States
WORD COUNT: 12386

ABSTRACT: The discovery of the adipose-derived hormone leptin has generated enormous interest in the interaction between peripheral signals and brain targets involved in the regulation of feeding and energy balance. Plasma leptin levels correlate with fat stores and respond to changes in energy balance. It was initially proposed that leptin serves a primary role as an anti-obesity hormone, but this role is commonly thwarted by leptin resistance. Leptin also serves as a mediator of the adaptation to fasting, and this role may be the primary function for which the molecule evolved. There is increasing evidence that leptin has systemic effects apart from those related to energy homeostasis, including regulation of neuroendocrine and immune function and a role in development. With permission, from the Annual Review of Physiology Volume 62, 2000, by Annual Reviews Inc. (http://www.annurev.org).

3/3,AB/48 (Item 1 from file: 156)
DIALOG(R)File 156:ToxFile

01165002 97133753 PMID: 8979148
Estriol: a potent regulator of TNF and IL-6 expression in a murine model of endotoxemia.
Zuckerman SH; Ahmari SE; Bryan-Poole N; Evans GF; Short L; Glasebrook AL

Division of Cardiovascular Research, Lilly Research Labs, Indianapolis, Indiana 46285, USA.
Inflammation (UNITED STATES) Dec 1996, 20 (6) p581-97, ISSN 0360-3997 Journal Code: GM0
Document type: Journal Article
Languages: ENGLISH

The increased incidence of autoimmune disease in *premenopausal"** women suggests the involvement of sex steroids in the pathogenesis of these disease processes. The effects of estrogen on autoimmunity and inflammation may involve changes in the secretion of inflammatory mediators by mononuclear phagocytes. Estradiol, for example, has been reported to regulate TNF, IL-6, *IL"**-*1"** and JE expression. In the present study the effects of the estrogen agonist, estriol, on cytokine expression have been investigated in mice administered a subletha1 lipopolysaccharide, LPS, challenge. Pretreatment of mice with pharmacologic doses of estriol, 0.4-2 mg/kg, resulted in a significant increase in serum TNF levels in both control and autoimmune MRL/lpr mice, following LPS challenge. This increase in TNF over the placebo group was blocked by the estrogen antagonist tamoxifen. Estriol treated mice also exhibited a rapid elevation in serum IL-6 levels following LPS challenge with the peak increase occurring 1 hr post LPS. This contrasted with the placebo group in which maximal serum IL-6 levels were detected at 3 hrs post challenge. This shift in the kinetics of IL-6 increase by estriol was inhibited by tamoxifen. The estriol mediated effects of TNF and IL-6 serum levels were consistent with the changes in TNF and IL-6 mRNA observed ex vivo in elicited peritoneal macrophages. Macrophage cultures from estriol treated animals however, did not demonstrate significant differences from the placebo group for TNF or NO secretion following in vitro LPS challenge. These results suggest that the estrogen agonist estriol can have significant quantitative, TNF, and kinetic, IL-6, effects on inflammatory monokines produced in response to an endotoxin challenge.

01833738 AADAAIC805829
Osteoprotegerin in bone metabolism
Author: Brandstrom, Helena Ingrid
Degree: Ph.D.
Year: 2001
Corporate Source/Institution: Uppsala Universitet (Sweden) (0903)
Source: VOLUME 62/03-C OF DISSERTATION ABSTRACTS INTERNATIONAL. PAGE 404. 56 PAGES
ISBN: 91-554-5016-4
Publisher: Uppsala University Library, Sweden

Bone turnover, remodeling, is a constant process replacing old bone with new. This complex cellular event involves resorption by osteoclasts and formation of new bone by osteoblasts. The balance between osteoblastic and osteoclastic activity is under regulation by several endocrine and paracrine factors. Osteoprotegerin (OPG), a recently discovered protein is an effective inhibitor of osteoclast formation and osteoclast activity. In this thesis, the regulation of OPG mRNA expression and protein secretion from human bone cells has been investigated. Also correlation between a single nucleotide *polymorphism"** in the OPG gene and bone mass has been studied. OPG mRNA levels were affected by several endocrine and paracrine

factors known to regulate bone resorption, such as prostaglandin E₂, TNFs, *interleukin"**-*1"** and glucocorticoid. An ELISA was developed and it was established that human osteoblasts secrete OPG protein and that the secretion is regulated by the above factors. A single nucleotide *polymorphism"** was discovered in the human OPG gene. There was no correlation between the *polymorphism"** and measures of bone mineral density in a cohort of 1044 post-*menopausal"** females. Surprisingly, a correlation between the *polymorphism"** and measures of vascular function and morphology was discovered. The data in the thesis show that OPG is produced in human bone marrow and is regulated by factors affecting the bone remodeling process, suggesting a central role for OPG in human bone turnover. The finding that the *polymorphism"** in the OPG gene correlates with vascular function opens up a new area of research aiming at understanding whether OPG might be involved also in cardiovascular diseases.

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 3/3,AB/50      (Item 1 from file: 65)
DIALOG(R)File  65:Inside Conferences
(c) 2002 BLDSC all rts. reserv. All rts. reserv.

03405329   INSIDE CONFERENCE ITEM ID: CN035948375
*IL"**-*1"** Receptor Antagonist Gene *Polymorphism"** Associated with Age
of  *Menopause"**
  van Dijk, S.; Stone, K. L.; Hannon, R. A.; Lui, L. L.; Sorrell,  J. A.;
Eastell, R.; Cummings, S. R.; Duff, G. W.
  CONFERENCE: American Society for Bone and Mineral Research-Annual
    meeting; 22nd
  JOURNAL OF BONE AND MINERAL RESEARCH, 2000; VOL 15; SUPPL 1  P: M341
  Blackwell Science, 2000
  ISSN: 0884-0431
  LANGUAGE: English  DOCUMENT TYPE: Conference Preprinted abstracts and
programme
    CONFERENCE SPONSOR: American Society for Bone and Mineral Research
    CONFERENCE LOCATION: Toronto, Canada
    CONFERENCE DATE:  Sep 2000

Set     Items   Description                                  - * Author(s)
S4        378   AU=(DUFF, G? OR DUFF G?)
S5         76   AU=(KORNMAN, K? OR KORNMAN K?)
S6         63   AU=(VAN DIJK, S? OR VAN DIJK S? OR VANDIJK, S? OR VANDIJK -
                  S?)
S7          0   S4 AND S5 AND S6
S8          5   S4 AND (S5 OR S6)
S9          1   S5 AND S6
S10         1   (S4 OR S5 OR S6) AND S1
S11         5   (S8 OR S9 OR S10) NOT S2
S12         5   RD (unique items)
>>>No matching display code(s) found in file(s): 65, 129, 158, 624

 12/3,AB/1      (Item 1 from file: 144)
DIALOG(R)File 144:Pascal
(c) 2002 INIST/CNRS. All rts. reserv.

  14606426   PASCAL No.: 00-0275153
  Effect of interleukin-1 gene polymorphisms on gingival inflammation
assessed by bleeding on probing in a periodontal maintenance population
  LANG N P; TONETTI M S; SUTER J; SORRELL J; *DUFF G W"**; *KORNMAN K S"**
```

Department of Periodontology and Fixed Prosthodontics, University of Berne, Switzerland; Department of Periodontology, Eastman Dental Institute, University College London, United Kingdom; Division of Molecular and Genetic Medicine, University of Sheffield, United Kingdom; Interleukin Genetics, San Antonio, Texas, United States

Journal: Journal of periodontal research, 2000, 35 (2) 102-107
Language: English

Bleeding on probing (BOP) is the most significant clinical parameter for the assessment of periodontal inflammation. The aim of this prospective longitudinal trial was to study the association between allelic variants of the IL-I gene complex and gingival inflammation. Three hundred and twenty-three randomly selected periodontal maintenance patients (64.4% females) received a periodontal examination that included probing depth measurements and BOP at each of 4 supportive periodontal therapy (SPT) appointments. A blood sample taken from each subject was analysed for the presence of specific allotypes of the IL-1 gene complex. Two polymorphisms located at + 4845 bp in the IL-1 alpha region and at + 3954 bp in the IL-1 beta region were evaluated by a polymerase chain reaction method; 35.3% of the examined subjects were positive for specific combinations of allotypes of the IL-1 gene complex previously associated with an increased risk for severe periodontitis. The population consisted of 90 current smokers and 94 former smokers. An analysis of the association between the IL-1 genotype and BOP in the whole population (including smokers) did not reach statistical significance because of the overriding effect of smoking. A subset analysis of the 139 never smokers indicated that genotype positive patients had a significantly elevated chance of presenting an increase in the BOP% over a 4-appointment recall period (p=0.03) after correcting for oral hygiene. In fact, patients who were genotype-negative had a 50% smaller chance of showing increases in BOP% during SPT. A further analysis explored the relationship between the genotype and the level of BOP% at the most recent recall visit. A generalized linear model showed a statistically significant effect of the genotype status after correcting for plaque accumulation and prevalence of residual pockets (>= 5 mm). Genotype-negative subjects had significantly lower BOP% (p=0.0097). It is concluded that the increased BOP prevalence and incidence observed in IL-1 genotype-positive subjects indicates that some individuals have a genetically determined hyper-inflammatory response that is expressed in the clinical response of the periodontal tissues.

12/3,AB/2 (Item 1 from file: 76)
DIALOG(R)File 76:Life Sciences Collection

02708052 5371725
A Sequence-Based Map of the Nine Genes of the Human Interleukin-1 Cluster
Nicklin, M.J.; Barton, J.L.; Nguyen, M.; Fitzgerald, M.G.; *Duff, G.W."**; *Kornman, K."**
Division of Genomic Medicine, University of Sheffield, Royal Hallamshire Hospital, Sheffield, S10 2JF, UK
Genomics vol. 79, no. 5, pp. 718-725 (2002)
ISSN: 0888-7543
DOCUMENT TYPE: Journal article LANGUAGE: ENGLISH
SUBFILE: Genetics Abstracts

Six novel genes encoding proteins with the interleukin (IL)-1 fold have been identified recently. The classical family members are involved in

inflammatory signaling. Previous work has placed the novel genes close to or within the same cluster as IL1A, IL1B, and IL1RN, which occupy an similar to 400-kb interval on chromosome 2. We have combined the incomplete public database sequence with our own sequence to generate a reference sequence and map that encompass all of the novel genes, allowing determination of the gene structures, precise localization of exons, and determination of distances between conventional SNP and microsatellite markers. Gene order from centromere to telomere is IL1A-IL1B-IL1F7-IL1F9-IL1F6-IL1F8-IL1F5-IL1F10-IL1RN, of which only IL1A, IL1B, and IL1F8 are transcribed towards the centromere. The gene order relates to the volutionary relationship between the genes. Key features of exon boundaries are conserved. There is no evidence for other IL-1 family members within the cluster. [copy]2002 Elsevier Science (USA).

12/3,AB/3 (Item 1 from file: 65)
DIALOG(R)File 65:Inside Conferences
(c) 2002 BLDSC all rts. reserv. All rts. reserv.

04052822 INSIDE CONFERENCE ITEM ID: CN042595834
Candidate Genes as Potential Links Between Periodontal and Cardiovascular Diseases
*Kornman, K. S."**; *Duff, G. W."**
CONFERENCE: Periodontal-systemeic connection: a state-of-the-science symposium
ANNALS OF PERIODONTOLOGY, 2001; VOL 6; NO 1 P: 48-57
American Academy of periodontology, 2001
LANGUAGE: English DOCUMENT TYPE: Conference Papers. described as proceedings
CONFERENCE SPONSOR: American Association of Periodontology
National Institute of Dental and Craniofacial Research
CONFERENCE LOCATION: Bethesda, MD 2001; Apr (200104) (200104)

12/3,AB/4 (Item 2 from file: 65)
DIALOG(R)File 65:Inside Conferences
(c) 2002 BLDSC all rts. reserv. All rts. reserv.

03122730 INSIDE CONFERENCE ITEM ID: CN033103270
Interleukin-1 genotypes and the association between periodontitis and cardiovascular disease
*Kornman, K. S."**; Pankow, J.; Offenbacher, S.; Beck, J.; di Giovine, F.; *Duff, G. W."**
CONFERENCE: Periodontal research-International conference; 11th
JOURNAL OF PERIODONTAL RESEARCH, 1999; VOL 34; NO 7 P: 353-357
Munksgaard, 1999
ISSN: 0022-3484
LANGUAGE: English DOCUMENT TYPE: Conference Papers
CONFERENCE LOCATION: Gothenburg, Sweden
CONFERENCE DATE: Jun 1999 (199906) (199906)

12/3,AB/5 (Item 3 from file: 65)
DIALOG(R)File 65:Inside Conferences
(c) 2002 BLDSC all rts. reserv. All rts. reserv.

02636424 INSIDE CONFERENCE ITEM ID: CN027455640
IL-1 Receptor Antagonist as a Potential New Therapeutic Agent for

Osteoporosis: A Computer Simulation Model of Bone Remodeling and Osteoporosis
Herren, L. T.; *Van Dijk, S."**; Wang, H. Y.; *Kornman, K. S."**
CONFERENCE: American Society for Bone and Mineral Research and International Bone and Mineral Society: ASBMR-IBMS second joint meeting-Joint meeting; 2nd
BONE -NEW YORK-, 1998; VOL 23; NUMB 5; SUPP 1 P: SA399
Elsevier Science, 1998
ISSN: 8756-3282
LANGUAGE: English DOCUMENT TYPE: Conference Preprinted abstracts and programme
CONFERENCE SPONSOR: American Society for Bone and Mineral Research International Bone and Mineral Society
CONFERENCE LOCATION: San Francisco, CA
CONFERENCE DATE: Dec 1998 (199812) (199812)
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18jul02 15:22:18 User219783 Session D1851.3

Myers
09/632657

Seq ID 5748

FILE 'REGISTRY' ENTERED AT 15:12:29 ON 18 JUL 2002
L1 43 SEA ABB=ON PLU=ON GCTGATATTCTGGTGGGAAA|GGCAAGAGCAAAACTC TGTC/SQSN

FILE 'HCAPLUS' ENTERED AT 15:17:30 ON 18 JUL 2002
L2 10 SEA ABB=ON PLU=ON L1

L2 ANSWER 1 OF 10 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:290495 HCAPLUS
Correction of: 2001:186025
DOCUMENT NUMBER: 134:350281
Correction of: 134:234030
TITLE: Gene expression marker nucleic acids and proteins for identification, assessment, prevention, and therapy of ovarian cancer
INVENTOR(S): Lee, John; Thompsho, Pamela; Lillie, James
PATENT ASSIGNEE(S): Millennium Predictive Medicine, Inc., USA
SOURCE: PCT Int. Appl., 1198 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001018542 A2		20010315	WO 2000-US24199	20000901

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG
RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG

PRIORITY APPLN. INFO.: US 1999-PV152547 19990903
US 2000-PV190347 20000316
US 2000-PV191321 20000321
US 2000-PV208382 20000531
US 2000-PV220467 20000720

AB The invention relates to compns., kits, and methods for detecting, characterizing, preventing, and treating human ovarian cancers. A variety of markers are provided, wherein changes in the levels of expression of one or more of the markers is correlated with the presence of ovarian cancer. The level of expression of the marker in a sample can be assessed, for example, by detecting the presence in the sample of: (1) a protein corresponding to the marker or a fragment of the protein using a reagent, such as an antibody or antibody deriv. or fragment, which binds specifically with the protein; (2) a transcribed polynucleotide (e.g., an mRNA or cDNA) having at least a portion with which the marker is substantially homologous by contacting a mixt. of transcribed polynucleotides obtained from the sample with a substrate having one or more of the markers provided; (3) a transcribed polynucleotide, wherein the polynucleotide anneals with the marker under stringent hybridization conditions. [This abstr. record is the second of five records for this document necessitated by the large no. of index entries required to fully index the document and publication system constraints.].

IT **202498-11-1**, GenBank AC004000

RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(nucleotide sequence; gene expression marker nucleic acids and proteins for identification, assessment, prevention, and therapy of ovarian cancer)

L2 ANSWER 2 OF 10 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:168192 HCAPLUS
DOCUMENT NUMBER: 134:217981
TITLE: Genetic techniques for determining genotype of IL-1 gene cluster (IL-1A, IL-1B and IL-1RN genes) in females, and their use in determining susceptibility of female to developing osteoporosis
INVENTOR(S): Van Dijk, Simon; Duff, Gordon W.
PATENT ASSIGNEE(S): Interleukin Genetics, Inc., USA
SOURCE: PCT Int. Appl., 68 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001016377	A2	20010308	WO 2000-US23844	20000830
WO 2001016377	A3	20020117		
W: AE, AU, BR, CA, CN, CZ, HU, IL, JP, KR, MX, NO, NZ, PL, RU, SG, TR, US, YU, ZA				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
BR 2000014150	A	20020514	BR 2000-14150	20000830
EP 1212464	A2	20020612	EP 2000-961425	20000830
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY				

PRIORITY APPLN. INFO.: US 1999-151460P P 19990830
WO 2000-US23844 W 20000830

AB The invention provides mol. genetic techniques for detg. the genotype of the IL-1 gene cluster (IL-1A, IL-1B and IL-1RN genes) in females, in order to det. the susceptibility of a female to developing osteoporosis. The invention relates that gene IL-1A encodes interleukin 1.alpha., gene IL-1B encodes interleukin 1.beta., while gene IL-1RN encodes interleukin 1 receptor antagonist. The invention provides that the IL-1A, IL-1B and IL-1RN alleles can be detecting using: (1) allele-specific hybridization; (2) DNA sequencing of a portion of the allele; (3) electrophoresis mobility of allele or fragments generated using a restriction endonuclease; (4) single-stranded conformation polymorphism; (5) oligonucleotide ligation assay, or (6) primer specific extension. The invention also relates that the alleles may be subjected to an amplification step prior to the detection steps listed above. The invention further relates that females contg. allele 2 of IL-1A, allele 2 of IL-1B (3954), allele 1 of IL-1B (-511), and allele 1 of IL-1RN (haplotype 1) are susceptible to larger bone loss and/or increased risk of fractures during the early menopausal years. Still further, the invention relates that females contg. allele 1 of IL-1A, allele 1 of IL-1B (3954), allele 2 of IL-1B (-511) and allele 2 of IL-1RN (haplotype 2) are susceptible to larger bone loss and/or

increased risk of fractures during post-menopause. The invention also provides a method for screening test compds. to identify therapeutics for osteoporosis, wherein the therapeutics are modulators (antagonists or agonists) of IL-1 activity. The invention further provides for the use of identified therapeutic in treatment and/or prevention of osteoporosis. Finally, the invention provides a method for detg. the effectiveness of therapeutic in a subject who has or is predisposed to developing osteoporosis.

IT **244295-41-8 244295-42-9**
RL: PRP (Properties)
(unclaimed sequence; genetic techniques for detg. genotype of IL-1 gene cluster (IL-1A, IL-1B and IL-1RN genes) in females, and their use in detg. susceptibility of female to developing osteoporosis)

L2 ANSWER 3 OF 10 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:12669 HCAPLUS
DOCUMENT NUMBER: 134:85136
TITLE: Diagnostics and therapeutics for diseases associated with an IL-1 inflammatory haplotype
INVENTOR(S): Duff, Gordon W.; Cox, Angela; Camp, Nicola Jane; Di, Giovine Francesco Saverio
PATENT ASSIGNEE(S): Interleukin Genetics, Inc., USA
SOURCE: PCT Int. Appl., 84 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001000880	A2	20010104	WO 2000-US18318	20000630
WO 2001000880	A3	20011115		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 6268142	B1	20010731	US 1999-345217	19990630
EP 1194590	A2	20020410	EP 2000-947005	20000630
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				

PRIORITY APPLN. INFO.:
US 1999-345217 A 19990630
GB 1997-11040 A 19970529
WO 1998-GB1481 A1 19980521
WO 2000-US18318 W 20000630

AB Methods and kits for detg. whether a subject has or is predisposed to developing a disease which is assocd. with IL-1 polymorphisms and assays for identifying therapeutics for treating and/or preventing the development of these diseases are provided. The disease of condition is selected from inflammatory disease, degenerative disease, immunol. disease, infectious disease, trauma induced disease, and cancer.

IT **244295-41-8 244295-42-9**
RL: ARG (Analytical reagent use); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(diagnostics and therapeutics for diseases assocd. with an IL-1 inflammatory haplotype)

L2 ANSWER 4 OF 10 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:842372 HCAPLUS
DOCUMENT NUMBER: 134:14046
TITLE: Diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family
INVENTOR(S): Francis, Sheila E.; Crossman, David C.; Duff, Gordon W.; Kornman, Kenneth S.
PATENT ASSIGNEE(S): Interleukin Genetics, Inc., USA
SOURCE: PCT Int. Appl., 122 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000072015	A2	20001130	WO 2000-US14775	20000526
WO 2000072015	A3	20020207		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1192279	A2	20020403	EP 2000-939389	20000526
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				

PRIORITY APPLN. INFO.: US 1999-320395 A 19990526
US 1999-431352 A 19991101
WO 2000-US14775 W 20000526

AB The kits and methods of the present invention relate to the diagnosis of cardiovascular disorders, ie., either a fragile plaque disorder, an occlusive disorder, or a restenosis disorder. Polymorphisms of the interleukin-1 gene family assocd. with cardiovascular disorders are identified as IL-1A(+4845), IL-1B(+3954), IL-1B(-511), IL-1RN(+2018), and alleles in linkage disequil. with these aforementioned alleles. Thus, predispositions toward cardiovascular disorders can be diagnosed by genotyping techniques such as size anal. of DNA fragments after restriction enzyme digestion. IL-1 genotypes are also correlated with target vessel revascularization, cholesterol levels, Lp(a) levels, LDL levels, and C-reactive protein levels. Other methods of the present invention relate to the selection of therapeutics for a patient with a cardiovascular disease.

IT **244295-41-8 244295-42-9**
RL: PRP (Properties)

(unclaimed nucleotide sequence; diagnostics and therapeutics for cardiovascular disorders based on genotyping of the interleukin-1 family)

L2 ANSWER 5 OF 10 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:842310 HCAPLUS
DOCUMENT NUMBER: 134:26093
TITLE: Diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family
INVENTOR(S): Kornman, Kenneth S.; Duff, Gordon W.; Crossman, David C.; Francis, Sheila E.; Stephenson, Katherine
PATENT ASSIGNEE(S): Interleukin Genetics, Inc., USA
SOURCE: PCT Int. Appl., 129 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000071753	A2	20001130	WO 2000-US14299	20000524
WO 2000071753	A3	20020207		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1192277	A2	20020403	EP 2000-937731	20000524
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				

PRIORITY APPLN. INFO.: US 1999-317674 A 19990524
US 1999-431352 A 19991101
WO 2000-US14299 W 20000524

AB Methods and kits for detg. whether a subject has or is predisposed to developing restenosis are provided. Polymorphisms of the interleukin-1 (IL-1) family assocd. with restenosis are identified as IL-1A(+4845), IL-1B(+3954), IL-1B(-511), IL-1RN(+2018), and IL-1RN(VNTR) or an allele that is in linkage disequil. with one of these alleles. Thus, predisposition to arterial restenosis can be diagnosed by genotyping techniques, such as size anal. of DNA fragments after restriction enzyme digestion. Such patients might be preferred candidates for surgical revascularization rather than percutaneous transluminal angioplasty, for example, or such patients may benefit from pharmacol. or topical interventions at an early stage that could affect the progression of the restenosis disorder.

IT **244295-41-8 244295-42-9**
RL: PRP (Properties)
(unclaimed nucleotide sequence; diagnostics and therapeutics for restenosis based on genotyping of the interleukin-1 family)

L2 ANSWER 6 OF 10 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:725801 HCAPLUS

DOCUMENT NUMBER: 133:294920
TITLE: Genetic markers for use in assessing the risk of interstitial lung disease
INVENTOR(S): Duff, Gordon W.; Di Giovine, Francesco Saverio; Whyte, Moria
PATENT ASSIGNEE(S): Interleukin Genetics, Inc., USA
SOURCE: PCT Int. Appl., 102 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000060117	A2	20001012	WO 2000-US8492	20000331
WO 2000060117	A3	20020207		
W: AE, AU, BR, CA, CN, CZ, HU, IL, JP, KR, MX, NO, NZ, PL, RU, SG, TR, US, YU, ZA				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
EP 1192275	A2	20020403	EP 2000-921536	20000331
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRIORITY APPLN. INFO.: US 1999-286108 A 19990402
WO 2000-US8492 W 20000331

AB The present invention provides novel methods and kits for detg. whether a subject has or is likely to develop an interstitial lung disorder such as pulmonary fibrosis; as well as methods for treating an ILD and screening assays for identifying novel ILD therapeutics. The method uses polymorphisms in the interleukin 1 gene cluster on 2q13 and the tumor necrosis factor region on chromosome 6 as indicators of risk.

IT **244295-41-8 244295-42-9**
RL: PRP (Properties)
(unclaimed nucleotide sequence; genetic markers for use in assessing the risk of interstitial lung disease)

L2 ANSWER 7 OF 10 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:441964 HCAPLUS
DOCUMENT NUMBER: 133:85082
TITLE: Diagnostic and therapeutics for sepsis by studying interleukin 1 gene polymorphism
INVENTOR(S): Di Giovine, Francesco S.; Duff, Gordon W.
PATENT ASSIGNEE(S): Interleukin Genetics, Inc., USA
SOURCE: PCT Int. Appl., 66 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000037679	A2	20000629	WO 1999-US25633	19991101
WO 2000037679	A3	20010111		
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU,				

ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 6251598 B1 20010626 US 1998-183850 19981030
EP 1127168 A2 20010829 EP 1999-972008 19991101
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO
US 2001034032 A1 20011025 US 2001-852948 20010510
PRIORITY APPLN. INFO.: US 1998-183850 A 19981030
WO 1999-US25633 W 19991101

AB Methods and kits for detecting polymorphism that are predictive of a subject's susceptibility to developing sepsis are described and sepsis therapeutics that address the mol. basis of the disease are described. The invention is based on detg. at least one allele of the interleukin 1 (IL-1) genetic pattern that leads to a dysregulated inflammatory response. In a further aspect, the invention features methods for treating or preventing the development of sepsis by administering an appropriate therapeutic of the invention.

IT **244295-41-8 244295-42-9**
RL: PRP (Properties)
(unclaimed nucleotide sequence; diagnostic and therapeutics for sepsis by studying interleukin 1 gene polymorphism)

L2 ANSWER 8 OF 10 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:366621 HCAPLUS
DOCUMENT NUMBER: 132:344010
TITLE: The DNA sequence of human chromosome 21
AUTHOR(S): Hattori, M.; Fujiyama, A.; Taylor, T. D.; Watanabe, H.; Yada, T.; Park, H.-S.; Toyoda, A.; Ishii, K.; Totoki, Y.; Choi, D.-K.; Soeda, E.; Ohki, M.; Takagi, T.; Sakaki, Y.; Taudlen, S.; Blechschmidt, K.; Polley, A.; Menzel, U.; Delabar, J.; Kumpf, K.; Lehmann, R.; Patterson, D.; Reichwald, K.; Rump, A.; Schillhabel, M.; Schudy, A.; Zimmermann, W.; Rosenthal, A.; Kudoh, J.; Shibuya, K.; Kawasaki, K.; Asakawa, S.; Shintani, A.; Sasaki, T.; Nagamine, K.; Mitsuyama, S.; Antonarakis, S. E.; Minoshima, S.; Shimizu, N.; Nordsiek, G.; Hornischer, K.; Brandt, P.; Scharfe, M.; Schon, O.; Desario, A.; Relchelt, J.; Kauer, G.; Blocker, H.; Ramser, J.; Beck, A.; Klages, S.; Hennig, S.; Riesselmann, L.; Dagand, E.; Haaf, T.; Wehrmeyer, S.; Borzym, K.; Gardiner, K.; Nizetic, D.; Francis, F.; Lehrach, H.; Reinhardt, R.; Yaspo, M.-L.
CORPORATE SOURCE: Genomic Sciences Center, RIKEN, Sagamihara, 228-8555, Japan
SOURCE: Nature (London) (2000), 405(6784), 311-319
CODEN: NATUAS; ISSN: 0028-0836
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Chromosome 21 is the smallest human autosome. An extra copy of chromosome 21 causes Down syndrome, the most frequent genetic cause of significant mental retardation, which affects up to 1 in 700 live births. Several anonymous loci for monogenic disorders and predispositions for common complex disorders have also been mapped to this chromosome, and loss of heterozygosity has been obsd. in regions assocd. with solid tumors. This report provides the sequence and gene catalog of the long arm of chromosome 21. At least 33,546,361 base pairs (bp) of DNA have been sequenced with very high accuracy, the largest contig being 25,491,867 bp. Only 3 small clone gaps and 7 sequencing gaps remain, comprising .apprx.100 kilobases. Thus, 99.7% coverage of 21q was achieved. About 281,116 bp were also sequenced from the short arm. The structural features identified include duplications that are probably involved in chromosomal abnormalities and repeat structures in the telomeric and pericentromeric regions. Anal. of the chromosome revealed 127 known genes, 98 predicted genes and 59 pseudogenes. The sequences are deposited in the GenBank database, and addnl. information can be sound from the home pages of the participating centers of the chromosome 21 sequencing consortium.

IT **264819-19-4**, GenBank AL163215
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(nucleotide sequence; DNA sequence of human chromosome 21)

REFERENCE COUNT: 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 9 OF 10 HCAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1999:691285 HCAPLUS
DOCUMENT NUMBER: 131:318551
TITLE: Fetal testing for prediction of low birth weight using PCR genotyping
INVENTOR(S): Kornman, Kenneth S.; Offenbacher, Steven; Duff, Gordon W.
PATENT ASSIGNEE(S): Medical Science Systems, Inc., USA
SOURCE: PCT Int. Appl., 78 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9954707	A2	19991028	WO 1999-US8794	19990421
WO 9954707	A3	20000622		
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2328955	AA	19991028	CA 1999-2328955	19990421
AU 9937557	A1	19991108	AU 1999-37557	19990421

EP 1071822 A2 20010131 EP 1999-919959 19990421
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI
JP 2002512047 T2 20020423 JP 2000-545003 19990421
PRIORITY APPLN. INFO.: US 1998-82487P P 19980421
WO 1999-US8794 W 19990421

AB Methods, assays and kits are disclosed for detecting a mother's or a fetus's susceptibility to an adverse pregnancy outcome such as low birth wt. The methods comprise obtaining a biol. sample from a patient and detg. the presence or absence of an IL-1 allele 2 of a marker that is assocd. with an adverse pregnancy outcome. Alleles for TNF.alpha. and Prostaglandin E2 were also genotyped. Detection of alleles by VNTR polymorphism and and PCR RFLP is described. Diagrams showing relationship between maternal urogenital or periodontal infection and fetal inflammatory response and maternal infection and inflammatory cytokines and risk of adverse pregnancy outcome. Genotype anal. was performed and characterized according to race from samples of maternal blood and chorionic villus and umbilical cord.

IT **244295-41-8 244295-42-9**
RL: PRP (Properties)
(unclaimed nucleotide sequence; fetal testing for prediction of low birth wt. using PCR genotyping)

L2 ANSWER 10 OF 10 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:641021 HCAPLUS
DOCUMENT NUMBER: 131:253337
TITLE: Methods and materials for the diagnosis of asthma wherein the genetic polymorphism D2S308*3 is the subject of detection
INVENTOR(S): Cookson, William Osmond Charles Michael; Moffatt, Miriam Fleur; Bhattacharyya, Sumit; Leaves, Nicholas
PATENT ASSIGNEE(S): Isis Innovation Ltd., UK
SOURCE: PCT Int. Appl., 21 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9950451	A1	19991007	WO 1999-GB968	19990326
W: AU, CA, JP, US				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9931587	A1	19991018	AU 1999-31587	19990326
EP 1064401	A1	20010103	EP 1999-913469	19990326
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRIORITY APPLN. INFO.: GB 1998-6652 A 19980327
WO 1999-GB968 W 19990326

AB A genetic method for diagnosing an individual as being asthmatic, or as having a predisposition to asthma, is described. The materials and methods of the invention are used to detect the presence or absence of the genetic polymorphism known as D2S308*3, which is located in a region of chromosome 2. The invention provides

oligonucleotide primers for use in detecting said polymorphism. The allele of identification may take the form of microsatellite repeats, and thus, oligonucleotide primers which hybridize to sequences at positions on either side of the microsatellite repeats are provided herein.

IT **244295-41-8 244295-42-9**
RL: PRP (Properties)
(unclaimed sequence; methods and materials for the diagnosis of asthma wherein the genetic polymorphism D2S308*3 is the subject of detection)

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

E1 THROUGH E4 ASSIGNED

FILE 'REGISTRY' ENTERED AT 15:18:39 ON 18 JUL 2002
L3 4 SEA FILE=REGISTRY ABB=ON PLU=ON (244295-41-8/BI OR 244295-42-9/BI OR 202498-11-1/BI OR 264819-19-4/BI)

L3 ANSWER 1 OF 4 REGISTRY COPYRIGHT 2002 ACS
RN **264819-19-4** REGISTRY
CN DNA (human clone R396A17 chromosome 21 segment HS21C015) (9CI) (CA INDEX NAME)
SQL 340000
MF Unspecified
CI MAN

REFERENCE 1: 132:344010

L3 ANSWER 2 OF 4 REGISTRY COPYRIGHT 2002 ACS
RN **244295-42-9** REGISTRY
CN PN: WO9950451 SEQID: 10 unclaimed sequence (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 14: PN: WO0037679 SEQID: 14 unclaimed DNA
CN 14: PN: WO0072015 SEQID: 17 unclaimed DNA
CN 18: PN: WO0071753 SEQID: 21 unclaimed DNA
CN 2: PN: WO0116377 PAGE: 22 unclaimed sequence
CN 4: PN: WO0060117 PAGE: 40 unclaimed DNA
CN 4: PN: WO0100880 SEQID: 7 claimed DNA
CN PN: WO9954707 SEQID: 22 unclaimed DNA
SQL 20 ← Seq. displayed @ L4 (2-4)
MF Unspecified
CI MAN

REFERENCE 1: 134:217981

REFERENCE 2: 134:85136

REFERENCE 3: 134:26093

REFERENCE 4: 134:14046

REFERENCE 5: 133:294920

REFERENCE 6: 133:85082

REFERENCE 7: 131:318551

REFERENCE 8: 131:253337

L3 ANSWER 3 OF 4 REGISTRY COPYRIGHT 2002 ACS
RN **244295-41-8** REGISTRY
CN PN: WO9950451 SEQID: 9 unclaimed sequence (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 13: PN: WO0037679 SEQID: 13 unclaimed DNA
CN 13: PN: WO0072015 SEQID: 16 unclaimed DNA
CN 17: PN: WO0071753 SEQID: 20 unclaimed DNA
CN 1: PN: WO0116377 PAGE: 22 unclaimed sequence
CN 3: PN: WO0060117 PAGE: 40 unclaimed DNA
CN 3: PN: WO0100880 SEQID: 6 claimed DNA
CN PN: WO9954707 SEQID: 21 unclaimed DNA
SQL 20 ← Seq. displayed C.L4 (3-4)
MF Unspecified
CI MAN

REFERENCE 1: 134:217981

REFERENCE 2: 134:85136

REFERENCE 3: 134:26093

REFERENCE 4: 134:14046

REFERENCE 5: 133:294920

REFERENCE 6: 133:85082

REFERENCE 7: 131:318551

REFERENCE 8: 131:253337

L3 ANSWER 4 OF 4 REGISTRY COPYRIGHT 2002 ACS
RN **202498-11-1** REGISTRY
CN DNA (human clone WO0118542_SEQID_3147 ovary tumor-associated protein cDNA) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN PN: WO0118542 SEQID: 3147 claimed DNA
SQL 128117
MF Unspecified
CI MAN

REFERENCE 1: 134:350281

L4 4 S L3 AND L1

L4 ANSWER 2 OF 4 REGISTRY COPYRIGHT 2002 ACS
RN **244295-42-9** REGISTRY
CN PN: WO9950451 SEQID: 10 unclaimed sequence (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 14: PN: WO0037679 SEQID: 14 unclaimed DNA
CN 14: PN: WO0072015 SEQID: 17 unclaimed DNA
CN 18: PN: WO0071753 SEQID: 21 unclaimed DNA
CN 2: PN: WO0116377 PAGE: 22 unclaimed sequence

CN 4: PN: WO0060117 PAGE: 40 unclaimed DNA
CN 4: PN: WO0100880 SEQID: 7 claimed DNA
CN PN: WO9954707 SEQID: 22 unclaimed DNA
CI MAN
SQL 20

SEQ 1 ggcaagagca aaactctgtc
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HITS AT: 1-20

REFERENCE 1: 134:217981

REFERENCE 2: 134:85136

REFERENCE 3: 134:26093

REFERENCE 4: 134:14046

REFERENCE 5: 133:294920

REFERENCE 6: 133:85082

REFERENCE 7: 131:318551

REFERENCE 8: 131:253337

L4 ANSWER 3 OF 4 REGISTRY COPYRIGHT 2002 ACS
RN **244295-41-8** REGISTRY
CN PN: WO9950451 SEQID: 9 unclaimed sequence (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 13: PN: WO0037679 SEQID: 13 unclaimed DNA
CN 13: PN: WO0072015 SEQID: 16 unclaimed DNA
CN 17: PN: WO0071753 SEQID: 20 unclaimed DNA
CN 1: PN: WO0116377 PAGE: 22 unclaimed sequence
CN 3: PN: WO0060117 PAGE: 40 unclaimed DNA
CN 3: PN: WO0100880 SEQID: 6 claimed DNA
CN PN: WO9954707 SEQID: 21 unclaimed DNA
CI MAN
SQL 20

SEQ 1 gctgatattc tggtgggaaa
========== ==========

HITS AT: 1-20

REFERENCE 1: 134:217981

REFERENCE 2: 134:85136

REFERENCE 3: 134:26093

REFERENCE 4: 134:14046

REFERENCE 5: 133:294920

REFERENCE 6: 133:85082

REFERENCE 7: 131:318551

REFERENCE 8: 131:253337

-key terms

```
=> e interleukin 1a/cn 5
E1            1     INTERLEUKIN 19 (HUMAN PRECURSOR)/CN
E2            1     INTERLEUKIN 19 (HUMAN)/CN
E3            0 --> INTERLEUKIN 1A/CN
E4            1     INTERLEUKIN 1H (HUMAN LONG ISOFORM A PRECURSOR)/CN
E5            1     INTERLEUKIN 1H (HUMAN LONG ISOFORM PRECURSOR)/CN

=> e "interleukin-1a"/cn 5
E1            1     INTERLEUKIN-18 RECEPTOR (HUMAN SUBUNIT ACPL)/CN
E2            1     INTERLEUKIN-18 RECEPTOR (HUMAN SUBUNIT IL-1RRP1)/CN
E3            0 --> INTERLEUKIN-1A/CN
E4            2     INTERLEUKIN-1B CONVERTING ENZYME (HUMAN ISOLATE 1 YEAR
                     OLD MALE CELL LINE THP.1 - ACUTE MONOCYTIC LEUKEMIA )
                    /CN
E5            1     INTERLEUKIN-1H1 (HUMAN KERATINOCYTE)/CN

=> s e4
L5            2 "INTERLEUKIN-1B CONVERTING ENZYME (HUMAN ISOLATE 1 YEAR
                OLD MALE CELL LINE THP.1 - ACUTE MONOCYTIC LEUKEMIA )"/CN

=> e interleukin 1rn/cn 5
E1            2     INTERLEUKIN 1H4 (HUMAN)/CN
E2            1     INTERLEUKIN 1HY2 (HUMAN SKIN GENE IL1HY2)/CN
E3            0 --> INTERLEUKIN 1RN/CN
E4            1     INTERLEUKIN 2 (107-THREONINE) (HUMAN)/CN
E5            1     INTERLEUKIN 2 (107-THREONINE,125-SERINE) (HUMAN)/CN

=> e "interleukin-1rn"/cn 5
E1            1     INTERLEUKIN-1H4 (HUMAN LUNG PRECURSOR)/CN
E2            1     INTERLEUKIN-1RA (DOG MONONUCLEAR CELL PRECURSOR)/CN
E3            0 --> INTERLEUKIN-1RN/CN
E4            1     INTERLEUKIN-2 (GUINEA PIG SPLENOCYTE PRECURSOR)/CN
E5            1     INTERLEUKIN-2 (HUMAN CLONE PILOT135-8)/CN

=> e il 1a/cn 5
E1            1     IL 11/CN
E2            1     IL 16/CN
E3            0 --> IL 1A/CN
E4            1     IL 2/CN
E5            1     IL 2066/CN

=> e "il-1a"/cn 5
E1            1     IL-17B RECEPTOR (HUMAN GENE IL17BR)/CN
E2            1     IL-19 (HUMAN CLONE HMQBM23 PRECURSOR)/CN
E3            0 --> IL-1A/CN
E4            1     IL-1RRP2 (HUMAN)/CN
E5            1     IL-1RRP2 (MOUSE)/CN

=> e "interleukin-1-.alpha."/cn 5
E1            1     INTERLEUKIN-1 RECEPTOR-TUMOR NECROSIS FACTOR RECEPTOR-
                    TUMOR NECROSIS FACTOR RECEPTOR FUSION PROTEIN (HUMAN)/
                    CN
E2            1     INTERLEUKIN-1 TYPE I RECEPTOR KINASE/CN
E3            0 --> INTERLEUKIN-1-.ALPHA./CN
E4            1     INTERLEUKIN-1-LIKE PROTEIN 1 (HUMAN CLONE PAC-131J6 GE
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                    NE IL1L1)/CN
E5          1       INTERLEUKIN-1-LIKE PROTEIN 1 (HUMAN PLACENTA GENE IL1L
                    1 TRANSCRIPT 2)/CN

=> e "interleukin 1-.alpha."/cn 5
E1          1       INTERLEUKIN 1 RECEPTOR-RELATED PROTEIN IL1-RRP1 (HUMAN
                     PRECURSOR)/CN
E2          1       INTERLEUKIN 1 RECEPTOR-RELATED PROTEIN IL1-RRP1 (MOUSE
                     PRECURSOR)/CN
E3          0 --> INTERLEUKIN 1-.ALPHA./CN
E4          1       INTERLEUKIN 1-BETA CONVERTASE (HUMAN GENE IL1BCE)/CN
E5          1       INTERLEUKIN 1-BETA CONVERTING ENZYME ISOFORM DELTA (HU
                    MAN CELL LINE THP-1 CLONE ICE-DELTA GENE IL1BCE)/CN

=> e "interleukin 1-alpha"/cn 5
E1          1       INTERLEUKIN 1 RECEPTOR-RELATED PROTEIN IL1-RRP1 (HUMAN
                     PRECURSOR)/CN
E2          1       INTERLEUKIN 1 RECEPTOR-RELATED PROTEIN IL1-RRP1 (MOUSE
                     PRECURSOR)/CN
E3          0 --> INTERLEUKIN 1-ALPHA/CN
E4          1       INTERLEUKIN 1-BETA CONVERTASE (HUMAN GENE IL1BCE)/CN
E5          1       INTERLEUKIN 1-BETA CONVERTING ENZYME ISOFORM DELTA (HU
                    MAN CELL LINE THP-1 CLONE ICE-DELTA GENE IL1BCE)/CN

=> e "interleukin 1alpha"/cn 5
E1          1       INTERLEUKIN 19 (HUMAN PRECURSOR)/CN
E2          1       INTERLEUKIN 19 (HUMAN)/CN
E3          0 --> INTERLEUKIN 1ALPHA/CN
E4          1       INTERLEUKIN 1H (HUMAN LONG ISOFORM A PRECURSOR)/CN
E5          1       INTERLEUKIN 1H (HUMAN LONG ISOFORM PRECURSOR)/CN

=> e interleukin 1/cn 5
E1          1       INTERLEUKIN (DOG STRAIN BEAGLE GENE IL-10 PRECURSOR)/C
                    N
E2          1       INTERLEUKIN (HUMAN CLONE PIL-1-14 REDUCED), N-L-METHIO
                    NYL-35-L-ASPARAGINE-/CN
E3          0 --> INTERLEUKIN 1/CN
E4          1       INTERLEUKIN 1 (FLOUNDER CLONE PSR.ALPHA.-IL2 PRECURSOR
                     REDUCED)/CN
E5          1       INTERLEUKIN 1 (FLOUNDER CLONE PSR.ALPHA.-IL2 REDUCED)/
                    CN

=> s interleukin 1 ?/cn
L6        101 INTERLEUKIN 1 ?/CN

=> s "interleukin-1"?/cn
L7        126 "INTERLEUKIN-1"?/CN

=> s l5 or l6 or l7
L8        216 L5 OR L6 OR L7

     FILE 'HCAPLUS' ENTERED AT 15:26:50 ON 18 JUL 2002
L9      39088 SEA ABB=ON  PLU=ON  L8 OR (INTERLEUKIN OR IL)(W)(1A OR 1
              OR 1B OR IA OR IB OR 1RN OR IRN) OR IL1A OR IL1B OR IL1A
L10        67 SEA ABB=ON  PLU=ON  L9 AND (PREMENOPAUS? OR PERIMENOPAUS?
               OR MENOPAUS? OR "CHANGE OF LIFE")
L11         4 SEA ABB=ON  PLU=ON  L10 AND (MUTAT? OR MUTANT OR
              MUTAGEN? OR POLYMORPH? OR POLY MORPH? OR (VARIAT? OR
```

VARIANT)(5A)ALLEL?)
L12 3 SEA ABB=ON PLU=ON L11 NOT L2

L12 ANSWER 1 OF 3 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:517497 HCAPLUS
DOCUMENT NUMBER: 134:99462
TITLE: Linkage of human tumor necrosis factor-alpha to human osteoporosis by sib-pair analysis
AUTHOR(S): Ota, N.; Hunt, S. C.; Nakajima, T.; Suzuki, T.; Hosoi, T.; Orimo, H.; Shirai, Y.; Emi, M.
CORPORATE SOURCE: Department of Molecular Biology, Institute of Gerontology, Nippon Medical School, Kawasaki, 211-8533, Japan
SOURCE: Genes and Immunity (2000), 1(4), 260-264
CODEN: GEIMA2; ISSN: 1466-4879
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Osteoporosis as well as osteopenia are common human conditions considered to result from the interplay of multiple genetic and environmental factors. Twin and family studies have yielded strong correlation between measures of bone mass and a no. of genetic factors. Certain genes (e.g., cytokines such as **interleukin -1**, interleukin-6, or tumor necrosis factor-alpha) are capable of regulating metab., formation, and resorption of bone; all processes that det. bone mass. We tested 192 sib-pairs of adult Japanese women from 136 families for genetic linkage between osteoporosis and osteopenia phenotypes and **allelic variants** at the tumor necrosis factor-alpha (TNFA) locus, using a dinucleotide-repeat **polymorphism** located near the gene. The TNFA locus showed evidence for linkage to osteoporosis, with mean allele sharing of 0.478 in discordant pairs and 0.637 in concordant affected pairs. Linkage with osteopenia was also significant in concordant affected pairs. Analyses limited to the post-**menopausal** women in our cohort showed similar or even stronger linkage for both phenotypes. The results provide evidence that genetic variations within the TNFA locus or adjacent genes affect regulation of mineral metab. in bone and some of them confer risk for osteoporosis in adult women.
REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 2 OF 3 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:653159 HCAPLUS
DOCUMENT NUMBER: 130:65022
TITLE: **Allelic variation** at the **interleukin-1** receptor antagonist gene is associated with early postmenopausal bone loss at the spine
AUTHOR(S): Keen, R. W.; Woodford-Richens, K. L.; Lanchbury, J. S.; Spector, T. D.
CORPORATE SOURCE: Twin & Genetic Epidemiology Research Unit, St. Thomas' Hospital, London, UK
SOURCE: Bone (New York) (1998), 23(4), 367-371
CODEN: BONEDL; ISSN: 8756-3282
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal

LANGUAGE: English

AB Genetic factors play an important role in detg. bone mineral d. (BMD) in later life, with the genetic influence mediated through effects on both peak mass and on age- and **menopause** -related bone loss. At **menopause** there is an increase in the prodn. and activity of various cytokines and growth factors within the bone microenvironment. The activity of **interleukin-1** (**IL-1**), a powerful stimulant of osteoclastic bone resorption, is increased in estrogen-deficient states with increased prodn. of **IL-1** and inhibition of the **IL-1** receptor antagonist (IL-1ra). Treatment with IL-1ra blocks the bone loss assocd. with ovariectomy in animals and the **IL-1** receptor antagonist gene (**IL-1RN**) is therefore a potential candidate gene for the regulation of postmenopausal bone loss. We examd. the relationship between annual rates of change in BMD over 5 yr and an 86 bp variable no. tandem-repeat **polymorphism** of the **IL-1RN** gene in 108 early postmenopausal women. All women were within 5 yr of a natural **menopause** at the study's onset, healthy, and not on hormone replacement therapy or other medication known to affect bone metab. BMD was measured annually over the 5 yr study period at the lumbar spine and femoral neck using dual-energy x-ray absorptiometry. Three alleles were identified (A1=4 repeats, A2=2 repeats, A3=5 repeats), with five genotypes obsd.: A1A1 (41.7%), A1A2 (45.4%), A2A2 (6.5%), A1A3 (2.8%), and A2A3 (3.7%). For anal., alleles were collapsed into a biallelic system grouping the A1 and A3 alleles. There was no significant relationship between the **IL-1RN** genotypes and baseline bone mass at either the spine or hip. **IL-1RN** genotype was significantly assocd. with annual rates of change in spinal bone mass ($p < 0.05$), and this finding remained significant after adjustment for age, wt., and baseline BMD. Carriage of at least one copy of the A2 allele was assocd. with reduced bone loss at the spine (mean change in BMD .+-. SD: -0.81.+-.1.46%/yr) when compared with noncarriage of the A2 allele (mean change -1.38.+-.1.48%/yr), $p = 0.05$. We therefore conclude that **allelic variation** at the **IL-1RN** locus is assocd. with differential rates of early postmenopausal bone loss at the spine. Further research will be required to clarify the mechanisms underlying these findings and to det. whether this assocn. translates into a significant long-term effect on BMD and fracture in later life.

REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 3 OF 3 HCAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1997:556335 HCAPLUS

DOCUMENT NUMBER: 127:229687

TITLE: Changes of bone metabolism with aging - in relation to involvement of estrogen withdrawal

AUTHOR(S): Gorai, Itsuo

CORPORATE SOURCE: Dep. Obstetrics Gynecol., Yokohama City Univ. Sch. Med., Yokohama, Japan

SOURCE: Nippon Sanka Fujinka Gakkai Zasshi (1997), 49(8), 537-545, 563-576
CODEN: NISFAY; ISSN: 0300-9165

PUBLISHER: Nippon Sanka Fujinka Gakkai

DOCUMENT TYPE: Journal; General Review
LANGUAGE: Japanese

AB A review and discussion with no refs. The diminution of bone mineral d. (BMD) in women is initiated at the onset of **menopause** and estrogen withdrawal causes rapid bone loss in early postmenopausal years, leading to an increase in a risk of osteoporotic fracture. Several cytokines are considered to be involved in the decrease of BMD due to estrogen deficiency. Recently, it has been reported that estrogen is important for bone maturation and mineralization in men as well as in women. In this study, we investigated the involvement of estrogen deficiency in bone metab. of postmenopausal women as compared with **premenopausal** women and assessed the factors relating to postmenopausal bone loss. In exptl. study, at 4 wk after ovariectomy (OVX), in OVX mice, femoral BMD decreased whereas the no. of bone marrow cells was greatly increased. To explore the endogenous bone-resorbing factors involved in estrogen deficiency, we examd. the bone-resorbing activity in the supernatant fraction of mouse bone marrow collected from ovariectomized mice. Only anti-interleukin (**IL**)-**1a** antibody completely neutralized the bone-resorbing activity in bone marrow supernatants from OVX mice. The concurrent addn. of **IL-1**, IL-6 sIL-6 receptor (R) and prostaglandin (PG)E2 co-operatively induced bone resorption. The effect of L-1a on collagenase and gelatinase activities and matrix metalloproteinase (MMP)-2 and MMP-13 mRNA expression was analyzed using **IL-1a** supplemented medium of organ culture of newborn mouse calvaria and osteoblasts from newborn mouse calvaria, resp. **IL-1a** increased collagenase and gelatinase activities as compared with those of control medium and stimulated MMP-2 and MMP-13 mRNA expression. Human osteoblast-like cells, HOS TE 85 cells, showed aromatase activity, which was detd. using 3H-androstenedione as a substrate and addn. of retinoic acid. TPA, bone morphogenic protein (BMP)-2 and dexamethasone increased their aromatase activity. As detd. by RT-PCR method, retinoic acid and TPA increased aromatase mRNA expression of HOS TE 85 cells. At 3 wk after orchidectomy (ORX), in ORX mice, the wt. of seminal vesicles and femoral BMD decreased whereas the no. of bone marrow cells was greatly increased. The change of hemopoiesis and bone metab. induced by ORX were completely restored by the treatment with 1 .mu.g of E2 but not with 10 .mu.g of DHT. In clin. study, several risk factors relating to low bone mass were assessed. Lumbar spine BMD significantly correlated with years since **menopause** (YSM) (r = -0.366), body wt. (r=0.350), age (r= -0.340), body mass index (BMI) (r=0.242), and height (r =0.212) in the postmenopausal women. In order to investigate an assocn. between vitamin D receptor (VDR) gene **polymorphism** and BMD or bone loss, we examd. VDR BsmI RFLP with amplification refractory **mutation** system (ARMS) and could not find any significant differences in lumbar spine baseline BMD between the bb genotype and the Bb genotype. In both early and late postmenopausal periods vertebral BMD with the Bb genotype decreased faster than that with the bb genotype (p=0.001). When we divided the subjects whose ages were from 45 to 55 yr into two subgroups (pre- and postmenopausal) to assess the effects of **menopause** on biochem. markers of bone resorption, we found a significant 110% increase in cross-linked N-telopeptides of type I collagen (NTx) and a 48% increase in lysylpyridinoline (LP) in post-**menopausal**

women compared with age-matched **premenopausal** women. The ROC anal. showed that the cutoff BMD values for discrimination of women with vertebral fracture varied according to the sites and methods of measurement ranging 59.0% of young adult mean value (YAM) of radial trabecular BMD by pQCT to 83.8% of young adult mean value (YAM) of femoral neck BMD by DXA.

(FILE 'MEDLINE, BIOSIS, EMBASE, WPIDS, CONFSCI, SCISEARCH, JICST-EPLUS, JAPIO' ENTERED AT 15:32:47 ON 18 JUL 2002)

L13 18 S L11

L14 14 DUP REM L13 (4 DUPLICATES REMOVED)

L14 ANSWER 1 OF 14 WPIDS (C) 2002 THOMSON DERWENT

ACCESSION NUMBER: 2002-010725 [01] WPIDS
DOC. NO. CPI: C2002-002607
TITLE: Detecting **polymorphisms** in the CAG and GGC repeats of the androgen receptor gene which are associated with bone mineral density is useful to determine a predisposition to osteoporosis.
DERWENT CLASS: B04 D16
INVENTOR(S): ROUSSEAU, F
PATENT ASSIGNEE(S): (SIGN-N) SIGNALGENE INC
COUNTRY COUNT: 95
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001073116	A2	20011004	(200201)*	EN	34

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW

AU 2001044004 A 20011008 (200208)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001073116	A2	WO 2001-CA402	20010328
AU 2001044004	A	AU 2001-44004	20010328

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001044004	A Based on	WO 200173116

PRIORITY APPLN. INFO: US 2000-192557P 20000328
AN 2002-010725 [01] WPIDS
AB WO 200173116 A UPAB: 20020105
NOVELTY - Determining predisposition to osteoporosis or low or high bone density or turnover comprising determining **polymorphisms** in the CAG and GGC repeats of the androgen receptor (AR) gene, is new.
DETAILED DESCRIPTION - Determining predisposition to

osteoporosis, low/high bone density/turnover and/or responsiveness to therapy for osteoporosis or prevention of low bone density, comprises determining androgen receptor (AR) **polymorphism** and analyzing **allelic variation** in a sample, where the AR **polymorphism** is (a) a CAG repeat and a GGC repeat **polymorphism**, or (b) an allele in linkage disequilibrium with (a).

INDEPENDENT CLAIMS are also included for the following:

(1) determining predisposition to low/high bone density, comprising determining androgen receptor **polymorphisms** in a sample, where a genotype showing a GGC repeat **polymorphism** associated with a CAG **polymorphism** can be correlated with high/low bone density or turnover;

(2) determining predisposition to androgen hormone-related medical conditions, comprising determining the length of a CAG and/or GGC repeat of the androgen receptor where length of the repeat is indicative of predisposition;

(3) using specific susceptible or resistant AR alleles to set up a screening assay for agents that modulate AR allele-specificity for the purpose preventing low bone density;

(4) selecting AR allele(s) suitable to screen for compounds that modulate AR activity, comprising constructing a recombinant AR comprising the allele(s), assaying function of the recombinant AR and selecting allele(s) that affect AR function;

(5) using combinations of AR **alleles** or their **variant, mutation** or equivalent which show linkage disequilibrium, to screen for agents that modulate AR function for the purpose of increasing bone density or potentiating action of an osteoporosis treatment;

(6) detecting an agent that modulates bone density, comprising contacting the agent with a cell comprising (a) an expression vector comprising a reporter operably linked to a promoter which has an AR response element which affects promoter activity upon binding to androgen, and (b) a combination of AR alleles and assaying the level of the reporter.

USE - The invention is used to determine predisposition to osteoporosis and to find new therapies for the condition.

Dwg.0/1

L14 ANSWER 2 OF 14 WPIDS (C) 2002 THOMSON DERWENT
ACCESSION NUMBER: 2001-265896 [27] WPIDS
DOC. NO. CPI: C2001-080455
TITLE: Determining whether a female is predisposed to the developing osteoporosis involves identifying **interleukin-1** haplotype pattern of the female, useful for selecting appropriate osteoporosis therapy .
DERWENT CLASS: B04 D16
INVENTOR(S): DUFF, G; VAN DIJK, S; DUFF, G W
PATENT ASSIGNEE(S): (INTE-N) INTERLEUKIN GENETICS INC
COUNTRY COUNT: 39
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001016377	A2	20010308	(200127)*	EN	69

RW: AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE
W: AE AU BR CA CN CZ HU IL JP KR MX NO NZ PL RU SG TR US YU ZA

AU 2000073380 A 20010326 (200137)
EP 1212464 A2 20020612 (200239) EN
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE
BR 2000014150 A 20020514 (200240)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001016377	A2	WO 2000-US23844	20000830
AU 2000073380	A	AU 2000-73380	20000830
EP 1212464	A2	EP 2000-961425	20000830
		WO 2000-US23844	20000830
BR 2000014150	A	BR 2000-14150	20000830
		WO 2000-US23844	20000830

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000073380	A	Based on	WO 200116377
EP 1212464	A2	Based on	WO 200116377
BR 2000014150	A	Based on	WO 200116377

PRIORITY APPLN. INFO: US 1999-151460P 19990830
AN 2001-265896 [27] WPIDS
AB WO 200116377 A UPAB: 20010518
NOVELTY - Determining (I) whether a female is predisposed to developing osteoporosis, involves identifying interleukin (**IL**)-**1** haplotype pattern (HP). Presence of HP 1 or HP 2 indicates that the female is susceptible to larger bone loss and/or increased risk of fracture during the early **menopausal** years (HP1) or post-**menopause** (HP2).

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for determining (II) the effectiveness of treating a subject that has or is predisposed to developing osteoporosis with a particular dose of a particular therapeutic comprising:

(a) detecting the level, amount or activity of an **IL-1** protein, or an **IL-1** mRNA or DNA in a sample obtained from a subject;

(b) administering the particular dose of the particular therapeutic to the subject, detecting the level, amount or activity of an **IL-1** protein or an **IL-1** mRNA or DNA in a sample obtained from the subject; and

(c) comparing the relative level, amount or activity of an **IL-1** protein obtained before and after the administration of the therapeutic.

USE - The method is useful for determining whether a female is predisposed to developing osteoporosis. (I) is useful for selecting an appropriate osteoporosis therapy for a female by performing (I) and selecting a therapeutic which is a modulator of an **IL-1** (preferably of **IL-1** alpha , **IL-1** beta , **IL-1RN**) activity (claimed).

The modulator is preferably a protein, peptide, peptidomimetic, small molecule, nucleic acid or a nutraceutical, agonist or an antagonist (claimed).

Information obtained from the assays is useful for determining whether a non-symptomatic subject has or is likely to develop the

particular disease or condition, and allows a more customized approach to preventing the onset or progression of the disease or condition and enables the clinician to effectively prescribe a therapy that addresses the molecular basis of the disease or condition. The detection of the alleles can indicate that the subject has or is predisposed to the development of osteoporosis. Dwg.0/5

L14 ANSWER 3 OF 14 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2001:526220 BIOSIS
DOCUMENT NUMBER: PREV200100526220
TITLE: **Polymorphisms** for interleukin-1beta (IL-1beta)-511 promoter, IL-1beta exon 5, and **IL-1** receptor antagonist: Nonassociation with endometriosis.
AUTHOR(S): Hsieh, Yao-Yuan; Chang, Chi-Chen; Tsai, Fuu-Jen (1); Wu, Jer-Yuarn; Shi, Yi-Ru; Tsai, Horng-Der; Tsai, Chang-Hai
CORPORATE SOURCE: (1) Department of Pediatrics and Medical Genetics, China Medical College Hospital, No. 2 Yuh-Der Road, Taichung: d0704@hpd.cmch.org.tw Taiwan
SOURCE: Journal of Assisted Reproduction and Genetics, (September, 2001) Vol. 18, No. 9, pp. 506-511. print. ISSN: 1058-0468.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Purpose: We aimed to investigate if interleukin-1beta (IL-1beta) and **IL-1** receptor antagonist (IL-1Ra) gene **polymorphism** could be used as markers of susceptibility in endometriosis. Materials and Methods: Women were divided into two groups: 1) endometriosis (n = 120); 2) nonendometriosis groups (n = 103). **Polymorphisms** for IL-1beta-511 promoter, IL-1beta exon 5, and IL-1Ra were detected by polymerase chain reaction. Genotypes and allelic frequencies for these **polymorphisms** in both groups were compared. Results: Proportions of different **IL-1** and IL-1Ra **polymorphisms** in both groups were nonsignificantly different. Proportions of C homozygote/heterozygote/T homozygote for IL-1beta-511 promoter in both groups were 1) 21.6/59.1/19.1% and 2) 26.2/50.5/23.3%. Proportions of E1 homozygote/heterozygote/E2 homozygote for IL-1beta exon 5 in both groups were 1) 91.6/5/3.3% and 2) 95.15/4.85/0%. Allele I/II/IV/V for IL-1Ra in both groups were 1) 92.5/5.4/1.6/0.4% and 2) 95.1/3.9/1/0%. Conclusions: Association of endometriosis with IL-1beta-511 promoter, IL-1beta exon 5, and **IL-1** receptor antagonist gene **polymorphisms** doesn't exist. These **polymorphisms** are not useful markers for prediction of endometriosis susceptibility.

L14 ANSWER 4 OF 14 JICST-EPlus COPYRIGHT 2002 JST
ACCESSION NUMBER: 1010689950 JICST-EPlus
TITLE: Etiology, Prevention and Treatment of Postmenopausal osteoporosis.
AUTHOR: KURABAYASHI TAKUMI
CORPORATE SOURCE: Niigatadai I Sankafujinkagakukyoshitsu
SOURCE: Niigata Igakkai Zasshi (Niigata Medical Journal), (2001) vol. 115, no. 4, pp. 122-126. Journal Code: F0877A

CODEN: NIGZAY; ISSN: 0029-0440
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Preprint
LANGUAGE: Japanese
STATUS: New

AB The etiology of postmenopausal osteoporosis has not completely been solved. Recent studies showed hypoestrogenic state caused the activation of cytokines (**IL-1**, IL-6, TNF-.ALPHA., GM-CSF etc.) from monocyte, stromal cells and osteoblast in bone marrow, and activated bone resorption and suppressed bone formation. Hormone replacement therapy (HRT) is the first choice of the treatment for postmenopausal osteoporosis. HRT is effective for not only osteoporosis but also climacteric disturbance and hyperlipidemia. HRT causes the increase of bone mineral density within 2 years, and mainteins it after 2 years. Vitamin D receptor gene **polymorphism** (Taq) is useful to detect the effect of HRT for osteoporosis. It is important for us to detect the high risk women for osteoporosis. Ovarian dysfunction, castration and GnRH agonist therapy etc. are risk factors for osteoporosis. Pregnancy and lactation may not be risk factors. There are two factors, i.e. genetic factor and environmental factor, to cause osteoporosis. The evaluation of genetic factor and the improvement of life style are very important to prevent osteoporosis. (author abst.)

L14 ANSWER 5 OF 14 WPIDS (C) 2002 THOMSON DERWENT
ACCESSION NUMBER: 2000-271467 [23] WPIDS
DOC. NO. CPI: C2000-082959
TITLE: Determination of susceptibility to, and/or response to therapy for osteoporosis used for increasing bone density comprises determining a combination of estrogen and vitamin D receptors **polymorphism.**
DERWENT CLASS: B04 D16
INVENTOR(S): ROUSSEAU, F
PATENT ASSIGNEE(S): (SIGN-N) SIGNALGENE INC
COUNTRY COUNT: 90
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000015836	A2	20000323	(200023)*	EN	49

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ TZ UG ZW
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW

AU 9957233	A	20000403	(200034)		
EP 1114181	A2	20010711	(200140)	EN	

R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000015836	A2	WO 1999-CA854	19990915

AU 9957233	A	AU 1999-57233	19990915
EP 1114181	A2	EP 1999-944190	19990915
		WO 1999-CA854	19990915

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9957233	A	Based on	WO 200015836
EP 1114181	A2	Based on	WO 200015836

PRIORITY APPLN. INFO: US 1998-100446P 19980915
AN 2000-271467 [23] WPIDS
AB WO 200015836 A UPAB: 20000516
NOVELTY - Determining susceptibility to, and/or response to therapy for osteoporosis of a human patient comprising determining a combination of estrogen and vitamin D receptors **polymorphism** or a **polymorphism** in linkage disequilibrium in a sample, is new.

DETAILED DESCRIPTION - Determining susceptibility to osteoporosis, and/or response to therapy for osteoporosis of a human patient comprises determining a combination of an estrogen receptor **polymorphism** or a **polymorphism** in linkage disequilibrium and a vitamin D receptor **polymorphism** or a **polymorphism** in linkage disequilibrium in a biological sample from the human patient where the estrogen **polymorphism** is selected from a PvuII **polymorphism** located in intron 1 of the estrogen receptor gene, or a DNA variant, equivalent or **mutation** which shows linkage disequilibrium with one of the allele of the PvuII **polymorphism**, and where the vitamin D receptor **polymorphism** is selected from a BsmI **polymorphism** located in intron 8 of the vitamin D receptor gene or a DNA variant, equivalent or **mutation** which shows linkage disequilibrium with one of the allele of the BsmI **polymorphism**.

INDEPENDENT CLAIMS are also included for the following:

(1) a prognosis kit for determining an outcome of an osteoporosis treatment or prevention program for a human patient comprising:

(a) at least one nucleic acid fragment specific for the estrogen receptor (ESR) where the fragment enables an assessment of the ESR **polymorphism** in intron 1 of the ESR gene; and

(b) at least one nucleic acid fragment specific for vitamin D receptor (VDR) where the fragment enables an assessment of the VDR **polymorphism** in intron 8 of the VDR gene; where a combination of **polymorphisms** at the ESR and VDR genes, or markers in linkage disequilibrium enables determination of the outcome of the treatment; and

(2) an assay for screening and selecting an agent which modulates bone density comprising:

(a) an expression vector comprising a promoter operably linked to a reporter gene, the promoter comprising estrogen receptor and vitamin D receptor response elements affecting the activity of the promoter upon binding of estrogen and vitamin D;

(b) a cell expressing a chosen allele of an estrogen receptor and a chosen allele of a vitamin D receptor, and harboring the vector of (a);

(c) submitting the cell to at least one agent; and

(d) assaying a level of the reporter gene, where an agent can be selected when the level of the reporter gene is significantly modulated by the presence of the agent.

USE - The method is useful for setting up a screening assay for agents destined to modulate estrogen receptor and/or vitamin D receptor function for the purpose of increasing bone density and/or potentiating an action of an osteoporosis treatment program.
Dwg.0/3

L14 ANSWER 6 OF 14 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2000:437912 BIOSIS
DOCUMENT NUMBER: PREV200000437912
TITLE: **IL-1** receptor antagonist gene **polymorphism** associated with age of **menopause**.
AUTHOR(S): van Dijk, S. (1); Stone, K. L.; Hannon, R. A.; Lui, L. L.; Sorrell, J. A.; Eastell, R.; Cummings, S. R.; Duff, G. W.
CORPORATE SOURCE: (1) Interleukin Genetics, Inc., San Antonio, TX USA
SOURCE: Journal of Bone and Mineral Research, (September, 2000) Vol. 15, No. Suppl. 1, pp. S537. print.
Meeting Info.: Twenty-Second Annual Meeting of the American Society for Bone and Mineral Research Toronto, Ontario, Canada September 22-26, 2000 American Society for Bone and Mineral Research
. ISSN: 0884-0431.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English

L14 ANSWER 7 OF 14 MEDLINE
ACCESSION NUMBER: 2000212592 MEDLINE
DOCUMENT NUMBER: 20212592 PubMed ID: 10750554
TITLE: Osteoporotic fractures are associated with an 86-base pair repeat **polymorphism** in the **interleukin-1**--receptor antagonist gene but not with **polymorphisms** in the interleukin-1beta gene.
AUTHOR: Langdahl B L; Lokke E; Carstens M; Stenkjaer L L; Eriksen E F
CORPORATE SOURCE: Department of Endocrinology and Metabolism, Aarhus University Hospital, Aarhus Amtssygehus, Denmark.
SOURCE: JOURNAL OF BONE AND MINERAL RESEARCH, (2000 Mar) 15 (3) 402-14.
Journal code: 8610640. ISSN: 0884-0431.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200006
ENTRY DATE: Entered STN: 20000629
Last Updated on STN: 20000629
Entered Medline: 20000619
AB Interleukin-1beta (IL-1beta) is a potent stimulator of bone resorption, and has been implicated in the pathogenesis of high bone turnover and osteoporosis. **IL-1** receptor antagonist (IL-1ra) is a competitive inhibitor of IL-1beta effects and the biological effects of IL-1beta are therefore proportional to

the ratio IL-1beta/IL-1ra. The coding regions of IL-1beta were examined for sequence variations by SSCP and sequencing after polymerase chain reaction (PCR) of genomic DNA. Three previously described **polymorphisms** (C(-511)-T, G(3877)-A and C(3954)-T) in the IL-1beta gene were determined by restriction fragment length **polymorphism** (RFLP) using Ava I, Aci I, and Taq I after PCR. The 86-base pair repeat **polymorphism** in IL-1ra was examined by PCR and electrophoresis and the T11100-C **polymorphism** in the IL-1ra gene was examined by RFLP using MspA1I after PCR. All **polymorphisms** were related to bone mass, biochemical markers of bone turnover, and presence of fracture in a study including 389 osteoporotic patients with vertebral fractures and normal controls. Two normal women were heterozygous for a shift from cytosine to thymine (C3263-T) in exon 4 of the IL-1beta gene. This substitution did not affect the amino acid sequence. We did not find other sequence variations in the IL-1beta gene apart from the already known **polymorphisms**. The distribution of C(-511)-T, G(3877)-A, and C(3954)-T genotypes was similar in the osteoporotic and the normal controls. No significant differences could be shown in bone mass or bone turnover. In the IL-1ra gene almost complete linkage was confirmed between the already known **polymorphisms**: G(1731)-A, G(1821)-A, A(1868)-G, G(1887)-C, T(8006)-C, C(8061)-T, 86 base pair variable number tandem repeat (VNTR), A(9589)-T, and a new **polymorphism**: T(1934)-C. The A1A1/A3 genotypes of the IL-1ra VNTR **polymorphism** were significantly more frequent in osteoporotic patients (56.2%) compared with age-matched normal controls (43.3%) (chi2 = 4.09; p = 0.043). The relative risk of osteoporotic fractures was increased to 1.68 (95% CI, 1.01-2.77) in individuals with A1A1/A3 genotypes. Bone mineral density (BMD) of the lumbar spine was reduced in individuals with A1A1/A3 genotypes (p = 0.014, analysis of variance [ANOVA]). The difference in bone mass between A1A1/A3 and A2A1/A2 tended to increase with increasing age. T1100-C genotypes were distributed similarly in osteoporotic patients and normal controls and the **polymorphism** was without effect on bone mass and biochemical markers of bone turnover. In conclusion, an 86-base pair repeat **polymorphism** in the IL-1ra gene is associated with increased risk of osteoporotic fractures. Other **polymorphisms** in the IL-1ra and the IL-1beta genes are not associated with osteoporotic fractures or alterations in bone mass or bone turnover.

L14 ANSWER 8 OF 14 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 2000:886548 SCISEARCH
THE GENUINE ARTICLE: 346YJ
TITLE: **IL-1** receptor antagonist gene **polymorphism** associated with age of **menopause.**
AUTHOR: vanDijk S (Reprint); Stone K L; Hannon R A; Lui L L; Sorrell J A; Eastell R; Cummings S R; Duff G W
CORPORATE SOURCE: INTERLEUKIN GENET INC, SAN ANTONIO, TX; UNIV CALIF SAN FRANCISCO, SAN FRANCISCO, CA 94143; UNIV SHEFFIELD, SHEFFIELD, S YORKSHIRE, ENGLAND
COUNTRY OF AUTHOR: USA; ENGLAND
SOURCE: JOURNAL OF BONE AND MINERAL RESEARCH, (SEP 2000) Vol. 15, Supp. [1], pp. M341-M341. Publisher: AMER SOC BONE & MINERAL RES, 2025 M ST, N W, STE 800, WASHINGTON, DC 20036-3309.

ISSN: 0884-0431.
DOCUMENT TYPE: Conference; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 0

L14 ANSWER 9 OF 14 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2001107468 MEDLINE
DOCUMENT NUMBER: 21040326 PubMed ID: 11196702
TITLE: Linkage of human tumor necrosis factor-alpha to human osteoporosis by sib pair analysis.
AUTHOR: Ota N; Hunt S C; Nakajima T; Suzuki T; Hosoi T; Orimo H; Shirai Y; Emi M
CORPORATE SOURCE: Department of Molecular Biology, Institute of Gerontology, Nippon Medical School, 1-396 Kosugi-cho, Nakahara-ku, Kawasaki 211-8533, Japan.
CONTRACT NUMBER: 1-P41-RR03655 (NCRR)
SOURCE: GENES AND IMMUNITY, (2000) 1 (4) 260-4.
Journal code: 100953417. ISSN: 1466-4879.
PUB. COUNTRY: England: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200102
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010208

AB Osteoporosis as well as osteopenia are common human conditions considered to result from the interplay of multiple genetic and environmental factors. Twin and family studies have yielded strong correlation between measures of bone mass and a number of genetic factors. Certain genes (e.g., cytokines such as **interleukin -1**, interleukin-6, or tumor necrosis factor-alpha) are capable of regulating metabolism, formation, and resorption of bone; all processes that determine bone mass. We tested 192 sib-pairs of adult Japanese women from 136 families for genetic linkage between osteoporosis and osteopenia phenotypes and **allelic variants** at the tumor necrosis factor-alpha (TNFA) locus, using a dinucleotide-repeat **polymorphism** located near the gene. The TNFA locus showed evidence for linkage to osteoporosis, with mean allele sharing of 0.478 (P = 0.30) in discordant pairs and 0.637 (P = 0.001) in concordant affected pairs. Linkage with osteopenia was also significant in concordant affected pairs (P = 0.017). Analyses limited to the post-**menopausal** women in our cohort showed similar or even stronger linkage for both phenotypes. The results provide evidence that genetic variations within the TNFA locus or adjacent genes affect regulation of mineral metabolism in bone and some of them confer risk for osteoporosis in adult women.

L14 ANSWER 10 OF 14 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 1998434113 MEDLINE
DOCUMENT NUMBER: 98434113 PubMed ID: 9763149
TITLE: **Allelic variation** at the **interleukin-1** receptor antagonist gene is associated with early postmenopausal bone loss at the spine.
AUTHOR: Keen R W; Woodford-Richens K L; Lanchbury J S;

Spector T D
CORPORATE SOURCE: Twin & Genetic Epidemiology Research Unit, St. Thomas' Hospital, London, UK.. r.keen@umds.ac.uk
SOURCE: BONE, (1998 Oct) 23 (4) 367-71.
Journal code: 8504048. ISSN: 8756-3282.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199812
ENTRY DATE: Entered STN: 19990115
Last Updated on STN: 19990115
Entered Medline: 19981204

AB Genetic factors play an important role in determining bone mineral density (BMD) in later life, with the genetic influence mediated through effects on both peak mass and on age- and **menopause** -related bone loss. At **menopause** there is an increase in the production and activity of various cytokines and growth factors within the bone microenvironment. The activity of **interleukin-1** (**IL-1**), a powerful stimulant of osteoclastic bone resorption, is increased in estrogen-deficient states with increased production of **IL-1** and inhibition of the **IL-1** receptor antagonist (IL-1ra). Treatment with IL-1ra blocks the bone loss associated with ovariectomy in animals and the **IL-1** receptor antagonist gene (**IL-1RN**) is therefore a potential candidate gene for the regulation of postmenopausal bone loss. We examined the relationship between annual rates of change in BMD over 5 years and an 86 bp variable number tandem-repeat **polymorphism** of the **IL-1RN** gene in 108 early postmenopausal women. All women were within 5 years of a natural **menopause** at the study's onset, healthy, and not on hormone replacement therapy or other medication known to affect bone metabolism. BMD was measured annually over the 5 year study period at the lumbar spine and femoral neck using dual-energy X-ray absorptiometry. Three alleles were identified (A1 = 4 repeats, A2 = 2 repeats, A3 = 5 repeats), with five genotypes observed: A1A1 (41.7%), A1A2 (45.4%), A2A2 (6.5%), A1A3 (2.8%), and A2A3 (3.7%). For analysis, alleles were collapsed into a biallelic system grouping the A1 and A3 alleles. There was no significant relationship between the **IL-1RN** genotypes and baseline bone mass at either the spine or hip. **IL-1RN** genotype was significantly associated with annual rates of change in spinal bone mass ($p < 0.05$), and this finding remained significant after adjustment for age, weight, and baseline BMD. Carriage of at least one copy of the A2 allele was associated with reduced bone loss at the spine (mean change in BMD +/- SD: -0.81 +/- 1.46%/year) when compared with noncarriage of the A2 allele (mean change -1.38 +/- 1.48%/year), $p = 0.05$. We therefore conclude that **allelic variation** at the **IL-1RN** locus is associated with differential rates of early postmenopausal bone loss at the spine. Further research will be required to clarify the mechanisms underlying these findings and to determine whether this association translates into a significant long-term effect on BMD and fracture in later life.

ACCESSION NUMBER: 97:685506 SCISEARCH
THE GENUINE ARTICLE: XP627
TITLE: Early **menopausal** bone loss at the spine is associated with **polymorphism** at the **interleukin** 1 receptor antagonist locus
AUTHOR: Keen R W (Reprint); WoodfordRichens K L; Major P J; Lanchbury J S; Spector T D
CORPORATE SOURCE: ST THOMAS HOSP, TWIN & OSTEOPOROSIS RES UNIT, LONDON, ENGLAND; UMDS, MOL IMMUNOGENET UNIT, LONDON, ENGLAND
COUNTRY OF AUTHOR: ENGLAND
SOURCE: JOURNAL OF BONE AND MINERAL RESEARCH, (AUG 1997) Vol. 12, Supp. [1], pp. T620-T620. Publisher: BLACKWELL SCIENCE INC, 350 MAIN ST, MALDEN, MA 02148. ISSN: 0884-0431.
DOCUMENT TYPE: Conference; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 0

L14 ANSWER 12 OF 14 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 97263849 EMBASE
DOCUMENT NUMBER: 1997263849
TITLE: Changes of bone metabolism with ageing in relation to involvement of estrogen withdrawal.
AUTHOR: Gorai I.
CORPORATE SOURCE: I. Gorai, Department of Obstetrics/Gynecology, Yokohama City Univ. School of Med., Yokohama, Japan
SOURCE: Acta Obstetrica et Gynaecologica Japonica, (1997) 49/8 (537-545). ISSN: 0300-9165 CODEN: AOGLAR
COUNTRY: Japan
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 003 Endocrinology
010 Obstetrics and Gynecology
020 Gerontology and Geriatrics
033 Orthopedic Surgery
LANGUAGE: Japanese
SUMMARY LANGUAGE: English; Japanese

AB The diminution of bone mineral density (BMD) in women is initiated at the onset of **menopause** and estrogen withdrawal causes rapid bone loss in early postmenopausal years, leading to an increase in a risk of osteoporotic fracture. Several cytokines are considered to be involved in the decrease of BMD due to estrogen deficiency. Recently, it has been reported that estrogen is important for bone maturation and mineralization in men as well as in women. In this study, we investigated the involvement of estrogen deficiency in bone metabolism of postmenopausal women as compared with **premenopausal** women and assessed the factors relating to postmenopausal bone loss. In experimental study, at 4 weeks after ovariectomy (OVX), in OVX mice, femoral BMD decreased whereas the number of bone marrow cells was greatly increased. To explore the endogenous bone-resorbing factors involved in estrogen deficiency, we examined the bone-resorbing activity in the supernatant fraction of mouse bone marrow collected from ovariectomized mice. Only anti-interleukin (**IL**)-1.alpha. antibody completely

neutralized the bone-resorbing activity in bone marrow supernatants from OVX mice. The concurrent addition of **IL-1**, IL-6, sIL-6 receptor (R) and prostaglandin (PG)E2 co-operatively induced bone resorption. The effect of L-2.alpha. on collagenase and gelatinase activities and matrix metalloproteinase (MMP)-2 and MMP-13 mRNA expression was analyzed using **IL-1** .alpha. supplemented medium of organ culture of newborn mouse calvaria and osteoblasts from newborn mouse calvaria, respectively. **IL-** 1.alpha. increased collagenase and gelatinase activities as compared with those of control medium and stimulated MMP-2 and MMP-13 mRNA expression. Human osteoblast-like cells, HOS TE 85 cells, showed aromatase activity, which was determined using 3H-androstenedione as a substrate and addition of retinoic acid, TPA, bone morphogenic protein (BMP)-2 and dexamethasone increased their aromatase activity. As determined by RT-PCR method, retinoic acid and TPA increased aromatase mRNA expression of HOS TE 85 cells. At 3 weeks after orchidectomy (ORX), in ORX mice, the weight of seminal vesicles and femoral BMD decreased whereas the number of bone marrow cells was greatly increased. The change of hemopoiesis and bone metabolism induced by ORX were completely restored by the treatment with 1 .mu.g of E2 but not with 10 .mu.g of DHT. In clinical study, several risk factors relating to low bone mass were assessed. Lumbar spine BMD significantly correlated with years since **menopause** (YSM) (r = - 0. 366), body weight (r = 0.350), age (r = -0.340), body mass index (BMI) (r =0.242), and height (r = 0.212) in the postmenopausal women. In order to investigate an association between vitamin D receptor (VDR) gene **polymorphism** and BMD or bone loss, we examined VDR Bsml RFLP with amplification refractory **mutation** system (ARMS) and could not find any significant differences in lumbar spine baseline BMD between the bb genotype and the Bb genotype. In both early and late postmenopausal periods vertebral BMD with the Bb genotype decreased faster than that with the bb genotype (p = 0.001). When we divided the subjects whose ages were from 45 to 55 years into two subgroups (pre- and postmenopausal) to assess the effects of **menopause** on biochemical markers of bone resorption, we found a significant 110% increase in crossdinked N- telopeptides of type I collagen (NTx) and a 48% increase in lysylpyridinoline (LP) in post-**menopausal** women compared with age-matched **premenopausal** women. The ROC analysis showed that the cutoff BMD values for discrimination of women with vertebral fracture varied according to the sites and methods of measurement ranging 59.0% of young adult mean value (YAM) of radial trabecular BMD by pQCT to 83.8% of young adult mean value (YAM) of femoral neck BMD by DXA.

L14 ANSWER 13 OF 14 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1997:239623 BIOSIS
DOCUMENT NUMBER: PREV19979538826
TITLE: Early **menopausal** bone loss at the spine is associated with **polymorphisms** at the **interleukin 1** receptor antagonist locus but not the interleukin 6 locus.
AUTHOR(S): Woodford-Richens, K. L. (1); Keen, R. W. (1); Lanchbury, J. S. (1); Spector, T. D.
CORPORATE SOURCE: (1) Mol. Immunogenet. Unit, UMDS, Guy's Hosp., London SE1 9RT UK
SOURCE: Bone (New York), (1997) Vol. 20, No. 4 SUPPL., pp.

8S.
Meeting Info.: 25th European Symposium on Calcified Tissues Harrogate, England, UK April 25-29, 1997
ISSN: 8756-3282.
DOCUMENT TYPE: Conference; Abstract
LANGUAGE: English

L14 ANSWER 14 OF 14 MEDLINE
ACCESSION NUMBER: 97133753 MEDLINE
DOCUMENT NUMBER: 97133753 PubMed ID: 8979148
TITLE: Estriol: a potent regulator of TNF and IL-6 expression in a murine model of endotoxemia.
AUTHOR: Zuckerman S H; Ahmari S E; Bryan-Poole N; Evans G F; Short L; Glasebrook A L
CORPORATE SOURCE: Division of Cardiovascular Research, Lilly Research Labs, Indianapolis, Indiana 46285, USA.
SOURCE: INFLAMMATION, (1996 Dec) 20 (6) 581-97.
Journal code: 7600105. ISSN: 0360-3997.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199704
ENTRY DATE: Entered STN: 19970414
Last Updated on STN: 19970414
Entered Medline: 19970402

AB The increased incidence of autoimmune disease in **premenopausal** women suggests the involvement of sex steroids in the pathogenesis of these disease processes. The effects of estrogen on autoimmunity and inflammation may involve changes in the secretion of inflammatory mediators by mononuclear phagocytes. Estradiol, for example, has been reported to regulate TNF, IL-6, **IL-1** and JE expression. In the present study the effects of the estrogen agonist, estriol, on cytokine expression have been investigated in mice administered a sublethal lipopolysaccharide, LPS, challenge. Pretreatment of mice with pharmacologic doses of estriol, 0.4-2 mg/kg, resulted in a significant increase in serum TNF levels in both control and autoimmune MRL/lpr mice, following LPS challenge. This increase in TNF over the placebo group was blocked by the estrogen antagonist tamoxifen. Estriol treated mice also exhibited a rapid elevation in serum IL-6 levels following LPS challenge with the peak increase occurring 1 hr post LPS. This contrasted with the placebo group in which maximal serum IL-6 levels were detected at 3 hrs post challenge. This shift in the kinetics of IL-6 increase by estriol was inhibited by tamoxifen. The estriol mediated effects of TNF and IL-6 serum levels were consistent with the changes in TNF and IL-6 mRNA observed ex vivo in elicited peritoneal macrophages. Macrophage cultures from estriol treated animals however, did not demonstrate significant differences from the placebo group for TNF or NO secretion following in vitro LPS challenge. These results suggest that the estrogen agonist estriol can have significant quantitative, TNF, and kinetic, IL-6, effects on inflammatory monokines produced in response to an endotoxin challenge.

(FILE 'MEDLINE' ENTERED AT 15:34:33 ON 18 JUL 2002)
L15 24511 SEA FILE=MEDLINE ABB=ON PLU=ON INTERLEUKIN-1/CT
L16 15086 SEA FILE=MEDLINE ABB=ON PLU=ON MENOPAUSE/CT

L17 2321 SEA FILE=MEDLINE ABB=ON PLU=ON PREMENOPAUSE/CT
L18 16 SEA FILE=MEDLINE ABB=ON PLU=ON L15 AND (L16 OR L17)

L18 ANSWER 1 OF 16 MEDLINE
AN 2001173753 MEDLINE
TI Interleukin-1beta-induced prostaglandin E2 production in human myometrial cells: role of a pertussis toxin-sensitive component.
AU Hertelendy F; Rastogi P; Molnar M; Romero R
SO AMERICAN JOURNAL OF REPRODUCTIVE IMMUNOLOGY, (2001 Mar) 45 (3) 142-7.
Journal code: 8912860. ISSN: 1046-7408.
AB PROBLEM: The objective of this study was to evaluate the possible role of pertussis toxin (PTX)-sensitive G-protein(s) in interleukin-1beta (IL-1) signaling in human myometrial cells (HMC). METHOD: Primary cultures of HMC were stimulated with human recombinant IL-1 alone or in combination with PTX. Prostaglandin (PG) E2 in the medium was measured by radioimmunoassay, cyclooxygenase type 2 (Cox-2) and IkappaB by western analysis, and the activities of two members of the mitogen-activated protein kinase (MAPK) family of enzymes, ERK-2 and JNK, by the phosphorylation of appropriate substrates. RESULTS: IL-1 increased PGE2 output during an 18-hr long incubation by 21.7-fold (n = 5 experiments). This increase was inhibited by 57% after pretreatment overnight with PTX. IL-1-induced expression of Cox-2 protein was also suppressed to a similar degree in PTX-treated HMC cultures. Degradation of the nuclear factor kappa B (NF-kappaB)-inhibiting protein (IkappaB), a critical step in IL-1 signaling to the nucleus, was significantly inhibited by PTX, as was IL-1-induced activation of ERK-2 and JNK. CONCLUSIONS: It is suggested that the occupied IL-1 receptor-generated signal in HMC is transmitted by multiple pathways. One is coupled to a PTX-sensitive G-protein upstream from the MAPK phosphorylation cascade. This, in turn, may interact with another signaling pathway, the activation of NF-kappaB, via the phosphorylation of the IkappaB kinase complex.

L18 ANSWER 2 OF 16 MEDLINE
AN 2000212592 MEDLINE
TI Osteoporotic fractures are associated with an 86-base pair repeat polymorphism in the interleukin-1--receptor antagonist gene but not with polymorphisms in the interleukin-1beta gene.
AU Langdahl B L; Lokke E; Carstens M; Stenkjaer L L; Eriksen E F
SO JOURNAL OF BONE AND MINERAL RESEARCH, (2000 Mar) 15 (3) 402-14.
Journal code: 8610640. ISSN: 0884-0431.
AB Interleukin-1beta (IL-1beta) is a potent stimulator of bone resorption, and has been implicated in the pathogenesis of high bone turnover and osteoporosis. IL-1 receptor antagonist (IL-1ra) is a competitive inhibitor of IL-1beta effects and the biological effects of IL-1beta are therefore proportional to the ratio IL-1beta/IL-1ra. The coding regions of IL-1beta were examined for sequence variations by SSCP and sequencing after polymerase chain reaction (PCR) of genomic DNA. Three previously described polymorphisms (C(-511)-T, G(3877)-A and C(3954)-T) in the IL-1beta gene were determined by restriction fragment length polymorphism (RFLP) using Ava I, Aci I, and Taq I after PCR. The 86-base pair repeat polymorphism in IL-1ra was examined by PCR and electrophoresis and the T11100-C polymorphism in the IL-1ra gene was examined by RFLP using MspAlI after PCR. All polymorphisms were related to bone mass, biochemical markers of bone turnover, and presence of fracture in a study

including 389 osteoporotic patients with vertebral fractures and normal controls. Two normal women were heterozygous for a shift from cytosine to thymine (C3263-T) in exon 4 of the IL-1beta gene. This substitution did not affect the amino acid sequence. We did not find other sequence variations in the IL-1beta gene apart from the already known polymorphisms. The distribution of C(-511)-T, G(3877)-A, and C(3954)-T genotypes was similar in the osteoporotic and the normal controls. No significant differences could be shown in bone mass or bone turnover. In the IL-1ra gene almost complete linkage was confirmed between the already known polymorphisms: G(1731)-A, G(1821)-A, A(1868)-G, G(1887)-C, T(8006)-C, C(8061)-T, 86 base pair variable number tandem repeat (VNTR), A(9589)-T, and a new polymorphism: T(1934)-C. The A1A1/A3 genotypes of the IL-1ra VNTR polymorphism were significantly more frequent in osteoporotic patients (56.2%) compared with age-matched normal controls (43.3%) (chi2 = 4.09; p = 0.043). The relative risk of osteoporotic fractures was increased to 1.68 (95% CI, 1.01-2.77) in individuals with A1A1/A3 genotypes. Bone mineral density (BMD) of the lumbar spine was reduced in individuals with A1A1/A3 genotypes (p = 0.014, analysis of variance [ANOVA]). The difference in bone mass between A1A1/A3 and A2A1/A2 tended to increase with increasing age. T1100-C genotypes were distributed similarly in osteoporotic patients and normal controls and the polymorphism was without effect on bone mass and biochemical markers of bone turnover. In conclusion, an 86-base pair repeat polymorphism in the IL-1ra gene is associated with increased risk of osteoporotic fractures. Other polymorphisms in the IL-1ra and the IL-1beta genes are not associated with osteoporotic fractures or alterations in bone mass or bone turnover.

L18 ANSWER 3 OF 16 MEDLINE
AN 2000141876 MEDLINE
TI Cytokine RNA levels in transiliac bone biopsies from healthy early postmenopausal women.
AU Abrahamsen B; Shalhoub V; Larson E K; Eriksen E F; Beck-Nielsen H; Marks S C Jr
SO BONE, (2000 Feb) 26 (2) 137-45.
Journal code: 8504048. ISSN: 8756-3282.
AB The cytokines interleukin-1beta (IL-1beta), tumor necrosis factor-alpha (TNF-alpha), and IL-6 induce osteoclast formation and may contribute to the development of postmenopausal osteoporosis. Cross-sectional studies have suggested that both IL-1 and IL-1ra secretion increase on estrogen withdrawal, and that postmenopausal osteoporosis is associated with an inadequate increase in monocyte IL-1ra secretion with age. We measured cytokine mRNA (IL-1beta, IL-1ra, IL-6, and TNF-alpha) directly in bone biopsies from early postmenopausal women to determine if a lower compensatory increase in IL-1ra mRNA could be demonstrated in women with rapid bone loss after the menopause. Biopsies were obtained from 23 early postmenopausal women (mean age 53.9 years) who participated in a randomized study of hormone replacement therapy (HRT) and risk factors for osteoporosis. Bone mineral density was assessed by duel energy X-ray absorptiometry at 0, 1, 2, and 5 years. Women in the control group were recruited to the biopsy study based on their observed rate of bone loss (upper or lower tertile). Consent was also obtained from 11 participants receiving HRT. Biopsies were taken at 2 years, frozen in nitrogen, and homogenized. Cytokine mRNA was measured by competitive reverse transcriptase polymerase chain reaction. The IL-1ra/IL-1beta mRNA slope for the slow-loss group was

steeper (deltaF = 23.3, p < 0.01) than that observed in the fast-loss group, indicating that slower bone loss was associated with higher IL-1ra mRNA levels relative to IL-1beta. During HRT, the IL-1beta mRNA level was inversely correlated with serum estradiol (log r2 = 0.77, p < 0.01), and women with a serum estradiol below 200 pmol/L during HRT had IL-1beta, mRNA levels identical to the control group. In contrast, IL-1ra mRNA was independent of serum estradiol. Histomorphometric analysis revealed weak correlations between IL-1beta mRNA and activation frequency (r2 = 0.26, p = 0.06) and between IL-1ra and volume referent bone resorption rate (r2 = 0.19, p = 0.11). TNF-alpha was not associated with the bone loss rates or with serum estradiol, and only three samples were positive for IL-6 mRNA. The findings support the hypothesis that IL-1beta production within bone increases with declining estrogen levels, and that an increase in II-1ra protects against accelerated bone loss.

L18 ANSWER 4 OF 16 MEDLINE
AN 2000009315 MEDLINE
TI Aging and cytokine production.
AU Pacifici R
SO CALCIFIED TISSUE INTERNATIONAL, (1999 Nov) 65 (5) 345-51.
Journal code: 7905481. ISSN: 0171-967X.

L18 ANSWER 5 OF 16 MEDLINE
AN 1999306464 MEDLINE
TI Bladder infection in the menopausal monkey.
AU Roberts J A; Kaack M B; Harrison R M; Klopp R; Ershler W
SO JOURNAL OF UROLOGY, (1999 Jul) 162 (1) 254-7.
Journal code: 0376374. ISSN: 0022-5347.
AB PURPOSE: The highest incidence of urinary tract infection in females occurs in elderly women. This study was done to determine whether this is due to the declining immune response that occurs during advancing age, or the menopausal state in the aged. MATERIALS AND METHODS: Adult female monkeys (average age 19 years) were studied, half being subjected to bilateral oophorectomy to produce the menopause. In addition, old females (average age 29 years) already at menopause were studied before and after hormonal replacement with estradiol and progesterone. Bacterial adherence to vaginal cells was studied prior to and after urethral infection with E. coli. Plasma estradiol and progesterone levels were done, as well as white blood counts, plasma cytokine assays and serum antibody titers. RESULTS: Bacteriuria was not prolonged, nor was there a significant difference in bacterial adherence to vaginal cells due to menopause. Interleukin-1 levels were depressed after surgical menopause but not as much as found in the old menopausal females and this low level was not corrected by hormonal replacement. The initial interleukin-2 levels were higher after spontaneous menopause, but the increasing plasma levels seen in cycling animals after infection did not occur in the aged menopausal females following infection even after hormone replacement. The antibody titers to the E. coli infection showed a trend to a lessened response to infection after menopause but were not significantly decreased. CONCLUSIONS: The deficient Il-1, Il-2 and antibody response following infection was not corrected by hormone replacement and thus appears to be due to aging rather than lack of female hormones. These facts may be explained by the T cell senescence known to occur in aged individuals.

L18 ANSWER 6 OF 16 MEDLINE

AN 1999036692 MEDLINE
TI Cytokine production and bone mineral density at the lumbar spine and femoral neck in premenopausal women.
AU Salamone L M; Whiteside T; Friberg D; Epstein R S; Kuller L H; Cauley J A
SO CALCIFIED TISSUE INTERNATIONAL, (1998 Dec) 63 (6) 466-70. Journal code: 7905481. ISSN: 0171-967X.
AB Cytokines such as interleukin-1 (IL-1beta), interleukin-6 (IL-6) and tumor necrosis factor (TNF-alpha) can influence both bone resorption and bone formation. The objective of this cross-sectional study was to examine the relationship between cytokine production by peripheral blood mononuclear cells (PBMC) and bone mineral density (BMD); the annual rate of change in BMD was examined. Subjects participating in a randomized clinical trial entitled the Women's Healthy Lifestyle Project in Allegheny County, Pennsylvania were used. They included 50 healthy premenopausal women, aged 45-52 years, who had regular menses within the past 3 months and were not on replacement estrogens. Dual-energy X-ray absorptiometry measurements at the AP lumbar spine and femoral neck were made at baseline and at the first annual exam using a Hologic QDR 2000 densitometer. Cytokine production of IL-1beta, IL-6, and TNF-alpha by PBMC was measured at the annual exam. The median values for stimulated cytokine production by PBMC were 3.92 ng/ml, 31.3 ng/ml, and 1.05 ng/ml, for IL-1beta, IL-6, and TNF-alpha, respectively. There were modest correlations between cytokine production and cross-sectional BMD, ranging from r = -0.30 to r = -0.13. Trends of greater spinal bone loss were observed in women with "high" (>/=75th percentile) cytokine production of stimulated IL-1beta and IL-6 (IL-1beta: "high" = -1.56% +/- 0.70 versus "low" (<75th percentile) = -0.56% +/- 0.35, P = 0.21). In contrast, greater annual gains in femoral neck BMD were observed in those with high cytokine production of IL-1beta and IL-6 (IL-1beta: high = 3.39% +/- 1.16 versus low = -0. 85 +/- 0.58, P = 0.002). There was no association between stimulated TNF production and annual change in BMD. In this population of healthy premenopausal women, the relationship between cytokine production by PBMC and the rate of change in BMD was significantly different for the lumbar spine and femoral neck, possibly reflecting differences in the proportion of trabecular and cortical bone at these sites.

L18 ANSWER 7 OF 16 MEDLINE
AN 95203486 MEDLINE
TI Stimulation of aromatase activity in breast fibroblasts by tumor necrosis factor alpha.
AU Macdiarmid F; Wang D; Duncan L J; Purohit A; Ghilchick M W; Reed M J
SO MOLECULAR AND CELLULAR ENDOCRINOLOGY, (1994 Dec) 106 (1-2) 17-21. Journal code: 7500844. ISSN: 0303-7207.
AB The conversion of androstenedione to estrone, the reaction mediated by the aromatase enzyme complex, may make an important contribution to the synthesis of estrogens in breast tissues. In the present study, the effect of the cytokine. TNF alpha, on aromatase activity was examined in breast fibroblasts derived from normal and malignant breast tissue. TNF alpha (2.5-10.0 ng/ml), in the presence of stripped fetal calf serum and dexamethasone, significantly stimulated fibroblast aromatase activity in a dose-dependent manner. IL-1 and IL-6 also stimulated fibroblast aromatase activity, but no marked synergism between TNF alpha and IL-1 or IL-6 was detected. Using a specific radioimmunoassay, significant concentrations of TNF

alpha were detected in samples of breast cyst fluid and breast tumor cytosol, which had previously been shown to stimulate aromatase activity, but not in conditioned medium from breast tumor-derived fibroblasts. As TNF alpha may be preferentially expressed and produced in the adipose tissue component of the breast, this cytokine may have an important role in regulating estrogen synthesis in normal and malignant breast tissues.

L18 ANSWER 8 OF 16 MEDLINE
AN 95067031 MEDLINE
TI Circulating levels of cytokines that modulate bone resorption: effects of age and menopause in women.
AU McKane W R; Khosla S; Peterson J M; Egan K; Riggs B L
SO JOURNAL OF BONE AND MINERAL RESEARCH, (1994 Aug) 9 (8) 1313-8.
Journal code: 8610640. ISSN: 0884-0431.
AB Interleukin 1 alpha (IL-1 alpha), interleukin 1 beta (IL-1 beta), and interleukin 6 (IL-6) are cytokines with potent bone-resorbing effects; some of these biologic effects are opposed by interleukin-1 receptor antagonist (IL-1ra). In vitro and animal model studies suggest that these cytokines are paracrine mediators of the increased bone resorption associated with estrogen deficiency, and increases in their production also could contribute to age-related bone loss. Therefore, we measured serum concentrations of these cytokines in 80 normal women who were 24-87 years old. IL-6 concentration correlated highly with age ($p < 0.001$) and increased three-fold during life. However, multiple-regression analysis showed no significant correlation between serum IL-6 levels and menopausal status, serum estradiol concentration, or markers for bone turnover (serum bone alkaline phosphatase, osteocalcin, carboxyl-terminal telopeptide of type I collagen, or 24 h urinary free pyridinoline excretion). Serum IL-1 alpha, IL-1 beta, or IL-1ra level did not change with age and, by multiple-regression analysis, did not correlate with markers of bone turnover, except IL-1ra weakly with ICTP. We found no relationship between bone-resorbing cytokines and ovarian function. Although the large age-related increase in serum IL-6 concentration could contribute to age-related bone loss, the lack of correlation with markers for bone turnover argues against this. However, based on the strong evidence in experimental animals that these cytokines are involved in estrogen action on bone, further studies in humans are warranted.

L18 ANSWER 9 OF 16 MEDLINE
AN 94358009 MEDLINE
TI Monocytic secretion of interleukin-1 receptor antagonist in normal and osteoporotic women: effects of menopause and estrogen/progesterone therapy.
AU Pacifici R; Vannice J L; Rifas L; Kimble R B
SO JOURNAL OF CLINICAL ENDOCRINOLOGY AND METABOLISM, (1993 Nov) 77 (5) 1135-41.
Journal code: 0375362. ISSN: 0021-972X.
AB Interleukin-1 (IL-1), a potent stimulant of bone resorption, has been implicated in the pathogenesis of postmenopausal osteoporosis, because monocyte IL-1 bioactivity increases after the menopause and is decreased by estrogen and progesterone (EP) replacement. As IL-1 bioactivity reflect the production of both IL-1 and the IL-1 inhibitor, IL-1 receptor antagonist (IL-1ra), EP treatment could decrease IL-1 bioactivity by regulating the secretion of either IL-1 or IL-1ra. We now report that EP treatment in vivo decreased the

secretion into the medium of cultured monocytes of IL-1ra and IL-1 beta as well as the IL-1 beta/IL-1ra ratio. We also found that in normal women the production of IL-1ra was within premenopausal levels in the first 7 yr after the menopause and increased linearly thereafter. In these women, monocyte IL-1 beta, IL-1 beta/IL-1ra ratio, and IL-1 bioactivity were all increased in the first 7 yr after the menopause and within the premenopausal range thereafter. In osteoporotic women, IL-1 beta, IL-1 beta/IL-1ra ratio, and IL-1 bioactivity increased after the menopause and returned to premenopausal levels after 15 yr from the menopause. In these women monocyte IL-1ra secretion was above the premenopausal range at all times after the menopause, but did not change with the passage of time since menopause. We conclude that hormone replacement decreases the in vitro secretion of both IL-1ra and IL-1 beta. The data also suggest that in normal women a progressive increase in the secretion of IL-1ra contributes to restore a normal IL-1/IL-1ra ratio after the menopause, a phenomenon which, in turn, may play a role in limiting postmenopausal bone loss.

L18 ANSWER 10 OF 16 MEDLINE
AN 94172090 MEDLINE
TI Gingival fluid IL-1 beta and IL-6 levels in menopause.
AU Reinhardt R A; Masada M P; Payne J B; Allison A C; DuBois L M
SO JOURNAL OF CLINICAL PERIODONTOLOGY, (1994 Jan) 21 (1) 22-5.
Journal code: 0425123. ISSN: 0303-6979.
AB Menopause and oophorectomy without estrogen therapy (ED) have been associated with increased production of bone-active cytokines by peripheral blood mononuclear cells. The current study extended evaluation to gingival crevicular fluid (GCF) levels of interleukin (IL)-1 beta and IL-6 in such subjects compared to premenopausal and postmenopausal estrogen-treated females (ES). 13 ED and 13 ES Caucasians with a history of moderate-severe adult periodontitis provided GCF from 1-3 clinically identical sites each (5-6 mm probing depth, 5-7 mm clinical attachment loss, bleeding on probing). 30 s GCF samples were obtained and evaluated for IL-1 beta and IL-6 levels using two-site enzyme-linked immunosorbent assays (ELISAs). The frequency of GCF IL-1 beta-positive subjects was elevated in ED versus ES (92% versus 23%; $p < 0.0004$, chi 2 analysis). IL-6 was detected more frequently in ED subjects (23% versus 8%; not significant); however, the frequency of IL-6 detection was low in both groups due to short sampling times. These data support the concept that clinical conditions causing low estrogen environments allow increased local production of the bone-active cytokine IL-1 beta, and perhaps IL-6.

L18 ANSWER 11 OF 16 MEDLINE
AN 94086760 MEDLINE
TI Peripheral monocyte culture supernatants of menopausal women can induce bone resorption: involvement of cytokines.
AU Cohen-Solal M E; Graulet A M; Denne M A; Gueris J; Baylink D; de Vernejoul M C
SO JOURNAL OF CLINICAL ENDOCRINOLOGY AND METABOLISM, (1993 Dec) 77 (6) 1648-53.
Journal code: 0375362. ISSN: 0021-972X.
AB Increased bone resorption is a mechanism contributing to bone loss in the postmenopausal period. Cytokines are involved in osteoclastic differentiation and, therefore, may play a role in the regulation of bone resorption. Several previous works showed the implication of

interleukin-1 (IL-1), IL-6, and tumor necrosis factor-alpha (TNF alpha) in the modulation of bone remodeling. This study determines the concomitant production of the three cytokines and tests the bone-resorbing activity of peripheral monocyte supernatants. Four groups of women were studied: premenopausal women (n = 13; mean age, 47 +/- 0.9 yr), untreated postmenopausal women (n = 21; mean age, 52 +/- 0.6 yr), postmenopausal women treated with estrogens (n = 14; mean age, 54.2 +/- 1.1 yr), or postmenopausal women treated with ethanehydroxydiphosphonate (n = 12; mean age, 53.2 +/- 2 yr). Assignment to clinical groups was verified by plasma FSH and estradiol determinations. Lumbar spine bone mineral density was significantly higher in the premenopausal women group than in the three postmenopausal groups. Peripheral blood monocytes were cultured for 48 h with 20% autologous plasma, and after stimulation with lipopolysaccharides. IL-1, IL-6, and TNF alpha levels were measured by RIA in the monocyte surpernatants. The three cytokines were highly correlated to each other, IL-1 with IL-6 ($r = 0.76$; $P < 0.001$), IL-1 with TNF alpha ($r = 0.89$; $P < 0.001$), and IL-6 with TNF alpha ($r = 0.89$; $P < 0.001$). The mean levels of the three cytokines could not be compared because of the variations in the values. However, a trend toward lower levels in the three cytokines was noted in estrogen-treated women compared to the untreated postmenopausals. The bone-resorbing activity of monocyte supernatants, assessed by fetal long bone-resorbing assay, increased in untreated postmenopausal compared to that in premenopausal women (1.22 +/- 0.08 vs. 0.87 +/- 0.11; $P < 0.05$). In estrogen-treated patients, this activity decreased to premenopausal levels (0.89 +/- 0.04 vs. 0.87 +/- 0.11; P = NS). The resorbing activity was correlated to IL-1 ($r = 0.28$; $P = 0.03$), IL-6 ($r = 0.52$; $P < 0.01$), and TNF alpha ($r = 0.48$; $P < 0.01$). The addition of cytokine inhibitors and IL-1 receptor antagonist and TNF alpha antibodies to the supernatant bone culture medium induced a significant decrease in the calcium release. Those data show the involvement of several cytokines in the bone resorption process after estrogen deficiency.

L18 ANSWER 12 OF 16 MEDLINE
AN 94076121 MEDLINE
TI Gingival crevicular fluid IL-8: correlation with local IL-1 beta levels and patient estrogen status.
AU Payne J B; Reinhardt R A; Masada M P; DuBois L M; Allison A C
SO JOURNAL OF PERIODONTAL RESEARCH, (1993 Nov) 28 (6 Pt 1) 451-3. Journal code: 0055107. ISSN: 0022-3484.
AB Gingival crevicular fluid (GCF) IL-8 and IL-1 beta levels were determined by sandwich enzyme-linked immunosorbent assays. Associations between IL-8 and IL-1 beta GCF levels, and between these cytokines and patient estrogen status were evaluated. IL-8 and IL-1 beta were detected more frequently and in higher amounts/30 s GCF sample in estrogen-deficient patients than in estrogen-sufficient patients. IL-8 and IL-1 beta GCF levels were significantly correlated. These findings suggest that GCF IL-8 levels are associated with patient estrogen status and local IL-1 beta concentrations.

L18 ANSWER 13 OF 16 MEDLINE
AN 94056531 MEDLINE
TI Interleukin-1 and tumor necrosis factor stimulate arachidonic acid release and phospholipid metabolism in human myometrial cells.
AU Molnar M; Romero R; Hertelendy F

SO AMERICAN JOURNAL OF OBSTETRICS AND GYNECOLOGY, (1993 Oct) 169 (4) 825-9.
Journal code: 0370476. ISSN: 0002-9378.

AB OBJECTIVE: Our aim was to evaluate the effects of the cytokines interleukin-1 and tumor necrosis factor on arachidonic acid release in human myometrial cells. STUDY DESIGN: Primary monolayer cultures of human myometrial cells prelabeled with tritiated arachidonic acid were exposed to interleukin-1 or tumor necrosis factor for varying periods and the release of tritiated arachidonic acid and its loss from phospholipids were measured by radiochromatography. To gain some information on the biologic action of interleukin-1 the contractile response to oxytocin was measured in myometrial strips preincubated with this cytokine. Data were statistically evaluated with analysis of variance or Student's test. RESULTS: Both cytokines caused a dose-dependent increase in tritiated arachidonate release that was suppressed by the protein synthesis inhibitor cycloheximide. Tritiated arachidonic acid release was maximal after 24 hours of stimulation with interleukin-1. Both interleukin-1 and tumor necrosis factor stimulated the release of the isotopically labeled fatty acid from phosphatidylcholine. In addition, interleukin-1 also increased the loss of arachidonic acid from phosphatidic acid and significantly potentiated the oxytocin-evoked myometrial contractility. CONCLUSIONS: Both interleukin-1 and tumor necrosis factor enhance arachidonic acid release, probably by inducing the synthesis of phospholipase A2 and possibly other enzymes involved in the metabolism of phospholipids. In turn, arachidonic acid itself may act as a second messenger, synergizing with other uterotonic agents, as well as serving as the precursor for prostaglandins and various other bioactive eicosanoids.

L18 ANSWER 14 OF 16 MEDLINE
AN 91031815 MEDLINE
TI The role of interleukin-1 in postmenopausal bone loss.
AU Pacifici R; Rifas L; McCracken R; Avioli L V
SO EXPERIMENTAL GERONTOLOGY, (1990) 25 (3-4) 309-16.
Journal code: 0047061. ISSN: 0531-5565.

AB Interleukin-1 (IL-1), a cytokine best known for its ability to stimulate lymphocyte proliferation, has recently been shown to stimulate bone resorption and modulate bone formation in vivo. Consequently, the authors have devised a series of studies to investigate the relationship between bone remodeling, menopause, and monocyte IL-1-secretion. In a first study, monocytes from osteoporotic patients were found to produce more IL-1 than monocytes from control subjects. IL-1 activity was also found to reflect histomorphometric indices of bone formation, but not of bone resorption. In a second study, devised to assess the effect of menopause on the relationship between IL-1 and bone turnover, a significant correlation was found between IL-1 and BGP in premenopausal osteoporotic women and osteoporotic men, but not in both postmenopausal osteoporotic subjects and normal subjects of either sex. In a third study, IL-1 from untreated postmenopausal women was found to be higher than in either untreated premenopausal or estrogen/progesterone-treated postmenopausal women. A significant negative correlation was found between IL-1 and years since menopause in both the healthy and osteoporotic postmenopausal women. Premenopausal IL-1 levels were achieved within eight years of menopause in the healthy but not in the osteoporotic subjects. In osteoporotic women, high IL-1 levels were evident as long as 15

years after menopause. IL-1 also correlated inversely with mineral density as measured by quantitative computer tomography. In prospective study, treatment with estrogen/progesterone caused a significant increase in IL-1 activity. This data indicates that monocyte IL-1 production mirrors the rate of bone turnover in both the healthy and osteoporotic patient, and that alteration in IL-1 production may underlie the postmenopausal acceleration of bone loss and its inhibition by ovarian steroids.

L18 ANSWER 15 OF 16 MEDLINE
AN 89184634 MEDLINE
TI Ovarian steroid treatment blocks a postmenopausal increase in blood monocyte interleukin 1 release.
AU Pacifici R; Rifas L; McCracken R; Vered I; McMurtry C; Avioli L V; Peck W A
SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1989 Apr) 86 (7) 2398-402.
Journal code: 7505876. ISSN: 0027-8424.
AB In previous studies, we showed that blood monocyte elaboration of interleukin 1 (IL-1), a known stimulator of bone resorption, was higher in osteoporotic patients with rapid bone turnover than in those with slow turnover and in nonosteoporotic subjects. Since an acceleration of bone loss following menopause contributes to the risk of osteoporosis in women, we have studied the effects of menopause and ovarian steroid treatment on IL-1 release by monocytes obtained from nonosteoporotic and osteoporotic women. IL-1 activity in the monocyte culture medium derived from untreated postmenopausal women (nonosteoporotic and osteoporotic) was higher than in the medium derived from either untreated premenopausal or estrogen/progesterone-treated postmenopausal women. A significant negative correlation was found between IL-1 and years since menopause in both the healthy ($r = -0.75$; P less than 0.005) and the osteoporotic ($r = -0.61$; P less than 0.01) untreated postmenopausal women. The difference between the two slopes was significant at P less than 0.05. Premenopausal IL-1 levels were achieved within 8 years of menopause in the nonosteoporotic, but not in the osteoporotic, subjects in whom increases were evident as long as 15 years after menopause. IL-1 also correlated inversely with vertebral mineral density ($r = -0.37$; P less than 0.05), as measured by quantitative computed tomography. In prospective studies, treatment with estrogen/progesterone for 1 month caused a substantial highly significant decrease in IL-1 activity in each of three nonosteoporotic and five osteoporotic women, confirming the apparent effect of hormone therapy observed in the cross-sectional analysis. Although a cause-effect relationship has not been established, it is our hypothesis, based on these data, that alterations in IL-1 production may underlie the postmenopausal acceleration in bone loss and its inhibition by ovarian steroids. Persistent elevation of IL-1 secretion appears to be a feature of postmenopausal osteoporosis.

L18 ANSWER 16 OF 16 MEDLINE
AN 89139743 MEDLINE
TI Effects of estrogen in vivo and in vitro on spontaneous interleukin-1 release by monocytes from postmenopausal women.
AU Stock J L; Coderre J A; McDonald B; Rosenwasser L J
SO JOURNAL OF CLINICAL ENDOCRINOLOGY AND METABOLISM, (1989 Feb) 68 (2) 364-8.
Journal code: 0375362. ISSN: 0021-972X.

AB Estrogen (E) inhibits bone resorption, but the mechanism of this effect is unknown. Interleukin-1 (IL-1) stimulates bone resorption in vitro and may be produced in bone by mononuclear phagocytes. Recently, the spontaneous release of IL-1 from peripheral monocytes was found to reflect bone formation in a subset of patients with idiopathic osteoporosis. We suspected that the action of E on bone is mediated indirectly by its effect on monocyte IL-1 activity. Eleven normal postmenopausal women taking no medications were given conjugated E (0.625 mg daily) for 3-9 weeks. Supernatants from cultured peripheral monocytes were analyzed for IL-1 production by stimulation of a cloned murine helper T-cell line. IL-1 release was expressed as a percentage of maximum release corrected for monocyte number. IL-1 release before E treatment was 11.0 +/- 0.2% (+/- SE), it was 7.8 +/- 1.6% after E treatment (P = NS). IL-1 release fell in each of the three women with the highest initial values (46% to 5%, 25% to 17%, and 18% to 12%). IL-1 release did not correlate with serum osteocalcin or fasting urinary calcium either before or after E treatment. Addition of 10(-7)-10(-10) mol/L 17 beta-estradiol to cultured monocytes obtained before E treatment caused an increase in IL-1 release that did not follow a dose-response relationship. Treatment of postmenopausal women with E did not affect spontaneous IL-1 release by peripheral monocytes in vitro. The addition of E in vitro did not produce consistent changes in IL-1 release by these cells. This does not exclude the possibility that E may affect monocyte IL-1 release in subsets of women with high spontaneous monocyte IL-1 release with or without osteoporosis.

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                 CONVERTING ENZYME (HUMAN ISOLATE 1 YEAR OLD MALE CELL
                 LINE THP.1 - ACUTE MONOCYTIC LEUKEMIA )"/CN
L6           101 SEA FILE=REGISTRY ABB=ON  PLU=ON  INTERLEUKIN 1 ?/CN
L7           126 SEA FILE=REGISTRY ABB=ON  PLU=ON  "INTERLEUKIN-1"?/CN
L8           216 SEA FILE=REGISTRY ABB=ON  PLU=ON  L5 OR L6 OR L7
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                 OR IB OR IBETA OR 1RN OR IRN) OR IL1A? OR IL1B? OR IL1A?
                 OR ILIB? OR IL1RN OR ILIRN OR IL1
L20          102 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L19 AND (PREMENOPAUS?
                 OR PERIMENOPAUS? OR MENOPAUS? OR "CHANGE OF LIFE")
L21            4 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L20 AND (MUTAT? OR
                 MUTANT OR MUTAGEN? OR POLYMORPH? OR POLY MORPH? OR
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=> s l21 not (l2 or l12)
L22            0 L21 NOT (L2 OR L12)

     (FILE 'MEDLINE, BIOSIS, EMBASE, WPIDS, CONFSCI, SCISEARCH,
     JICST-EPLUS, JAPIO' ENTERED AT 15:38:34 ON 18 JUL 2002)
L23           20 S L21
L24            2 S L23 NOT L13
L25            2 DUP REM L24 (0 DUPLICATES REMOVED)

L25  ANSWER 1 OF 2  SCISEARCH  COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER:    2001:247140  SCISEARCH
THE GENUINE ARTICLE: 409VA
TITLE:               Sex steroids and bone
AUTHOR:              Compston J E (Reprint)
```

CORPORATE SOURCE: Addenbrookes Hosp, Dept Med, Level 5, Box 157, Cambridge CB2 2QQ, England (Reprint); Univ Cambridge, Sch Clin Med, Dept Med, Cambridge, England
COUNTRY OF AUTHOR: England
SOURCE: PHYSIOLOGICAL REVIEWS, (JAN 2001) Vol. 81, No. 1, pp. 419-447.
Publisher: AMER PHYSIOLOGICAL SOC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814 USA.
ISSN: 0031-9333.
DOCUMENT TYPE: General Review; Journal
LANGUAGE: English
REFERENCE COUNT: 417

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Sex steroids are essential for skeletal development and the maintenance of bone health throughout adult life, and estrogen deficiency at **menopause** is a major pathogenetic factor in the development of osteoporosis in postmenopausal women. The mechanisms by which the skeletal effects of sex steroids are mediated remain incompletely understood, but in recent years there have been considerable advances in our knowledge of how estrogens and, to a lesser extent androgens, influence bone modeling and remodeling in health and disease. New insights into estrogen receptor structure and function, recent discoveries about the development and activity of osteoclasts, and lessons learned from human and animal genetic **mutations** have all contributed to increased understanding of the skeletal effects of estrogen, both in males and females. Studies of untreated and treated osteoporosis in postmenopausal women have also contributed to this knowledge and have provided unequivocal evidence for the potential of high-dose estrogen therapy to have anabolic skeletal effects. The development of selective estrogen receptor modulators has provided a new approach to the prevention of osteoporosis and other major diseases of **menopause** and has implications for the therapeutic use of other steroid hormones, including androgens. Further elucidation of the mechanisms by which sex steroids affect bone thus has the potential to improve the clinical management not only of osteoporosis, both in men and women, but also of a number of other diseases related to sex hormone status.

L25 ANSWER 2 OF 2 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 96:779075 SCISEARCH
THE GENUINE ARTICLE: VN326
TITLE: INFLUENCE OF VITAMIN-D-RECEPTOR GENOTYPE ON BONE MASS CHANGES AFTER RENAL-TRANSPLANTATION
AUTHOR: TORRES A (Reprint); MACHADO M; CONCEPCION M T; MARTIN N; LORENZO V; HERNANDEZ D; RODRIGUEZ A P; RODRIGUEZ A; DEBONIS E; GONZALEZPOSADA J M; HERNANDEZ A; SALIDO E
CORPORATE SOURCE: UNIV HOSP, SERV NEFROL, OFRA S-N, LA LAGUNA 38320, TENERIFE, SPAIN (Reprint); UNIV LA LAGUNA, HOSP UNIV CANARIAS, SERV NEFROL, TENERIFE, SPAIN; UNIV LA LAGUNA, HOSP UNIV CANARIAS, SERV RADIODIAGNOST, TENERIFE, SPAIN; UNIV LA LAGUNA, HOSP UNIV CANARIAS, UNIDAD INVEST, TENERIFE, SPAIN
COUNTRY OF AUTHOR: SPAIN
SOURCE: KIDNEY INTERNATIONAL, (NOV 1996) Vol. 50, No. 5, pp. 1726-1733.

ISSN: 0085-2538.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE; CLIN
LANGUAGE: ENGLISH
REFERENCE COUNT: 37

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Renal transplant patients immunosuppressed with cyclosporine A (CsA) exhibit both a significant bone loss and an increased rate of bone fractures. An association between common **allelic variants** of the the vitamin D receptor (VDR) gene and bone mineral density and turnover has been reported in adults. However, the genetic influence on the rate of bone loss after renal transplantation has not been explored. We prospectively determined the changes in spinal mineral density in 34 consecutive nondiabetic adults who received a cadaveric renal allograft. Serum biochemical markers of bone metabolism and the vertebral mineral density (VMD) assessed by quantitative computed tomography were determined at the time of transplantation and three and twelve months later. In fifteen patients the histomorphometric features of **iliac** bone were analyzed at baseline and twelve months after transplantation. VDR alleles were typed by a PCR assay based on a **polymorphic** BsmI restriction site. Patients with the so-called ''favorable'' bb genotype (N = 12) were compared with those with the Bb or BB genotype (N = 22). Baseline VMD was similar in patients with or without the favorable bb genotype. Three months after transplantation the mean (+/- SD) VMD decreased 14 +/- 13.3 percent in all patients (16.5 +/- 13.1% in patients homozygous for the b allele and 13.77 +/- 13.9% in those with Bb or BB genotypes). The rate of VMD loss at this time inversely correlated with pretransplant PTH levels ($r = -0.40$; $P < 0.05$). Between 3 and 12 months after transplantation, patients with the favorable bb genotype recovered more VMD than those with Bb or BB types and showed a significantly higher Z score at the end of the follow-up (-0.37 +/- 1.16 vs. -1.10 +/- 1.20, respectively: $P < 0.05$). The beneficial effect of bb genotype was independent of the prevailing PTH levels and was also observed in those patients with a baseline PTH level < 250 pg/ml (final Z score: bb, -0.42 +/- 1.3, N = 11; Bb/BB, -1.35 +/- 0.8, N = 11, $P < 0.05$). At the end of follow-up, the histomorphometric studies shelved a higher bone formation rate adjusted for PTH levels in patients with the Bb or BB genotype than in those with the favorable bb genotype (0.29 +/- 0.06 vs. 0.21 +/- 0.08 mu m(3)/mu m(2)/day respectively; $P < 0.05$). In conclusion, high pretransplant PTH levels enhance the early trabecular bone loss after renal transplantation, and functionally different alleles of the vitamin D receptor gene may condition the bone turnover and the degree of recovery of the bone mass.

(FILE 'HCAPLUS, MEDLINE, BIOSIS, EMBASE, WPIDS, CONFSCI, SCISEARCH, JICST-EPLUS, JAPIO' ENTERED AT 15:40:35 ON 18 JUL 2002)

L26 1414 S DUFF G?/AU
L27 501 S KORNMAN K?/AU
L28 147 S VAN DIJK S?/AU
L29 0 S L26 AND L27 AND L28
L30 42 S L26 AND (L27 OR L28)
L31 0 S L27 AND L28
L32 4 S (L30 OR L26 OR L27 OR L28) AND L20
L33 3 DUP REM L32 (1 DUPLICATE REMOVED)

- Author(s)

L33 ANSWER 1 OF 3 HCAPLUS COPYRIGHT 2002 ACS DUPLICATE 1
ACCESSION NUMBER: 2001:168192 HCAPLUS
DOCUMENT NUMBER: 134:217981
TITLE: Genetic techniques for determining genotype of **IL-1** gene cluster (**IL-1A, IL-1B** and **IL-1RN** genes) in females, and their use in determining susceptibility of female to developing osteoporosis
INVENTOR(S): **Van Dijk, Simon; Duff, Gordon W.**
PATENT ASSIGNEE(S): Interleukin Genetics, Inc., USA
SOURCE: PCT Int. Appl., 68 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001016377	A2	20010308	WO 2000-US23844	20000830
WO 2001016377	A3	20020117		
W: AE, AU, BR, CA, CN, CZ, HU, IL, JP, KR, MX, NO, NZ, PL, RU, SG, TR, US, YU, ZA				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
BR 2000014150	A	20020514	BR 2000-14150	20000830
EP 1212464	A2	20020612	EP 2000-961425	20000830
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY				

PRIORITY APPLN. INFO.: US 1999-151460P P 19990830
WO 2000-US23844 W 20000830

AB The invention provides mol. genetic techniques for detg. the genotype of the **IL-1** gene cluster (**IL-1A, IL-1B** and **IL-1RN** genes) in females, in order to det. the susceptibility of a female to developing osteoporosis. The invention relates that gene **IL-1A** encodes **interleukin 1**.alpha., gene **IL-1B** encodes **interleukin 1**.beta., while gene **IL-1RN** encodes **interleukin 1** receptor antagonist. The invention provides that the **IL-1A, IL-1B** and **IL-1RN** alleles can be detecting using: (1) allele-specific hybridization; (2) DNA sequencing of a portion of the allele; (3) electrophoresis mobility of allele or fragments generated using a restriction endonuclease; (4) single-stranded conformation polymorphism; (5) oligonucleotide ligation assay, or (6) primer specific extension. The invention also relates that the alleles may be subjected to an amplification step prior to the detection steps listed above. The invention further relates that females contg. allele 2 of **IL-1A**, allele 2 of **IL-1B** (3954), allele 1 of **IL-1B** (-511), and allele 1 of **IL-1RN** (haplotype 1) are susceptible to larger bone loss and/or increased risk of fractures during the early **menopausal** years. Still further, the invention relates that females contg. allele 1 of **IL-1A**, allele 1 of **IL-1B** (3954), allele 2

of **IL-1B** (-511) and allele 2 of **IL-1RN** (haplotype 2) are susceptible to larger bone loss and/or increased risk of fractures during post-**menopause**. The invention also provides a method for screening test compds. to identify therapeutics for osteoporosis, wherein the therapeutics are modulators (antagonists or agonists) of **IL-1** activity. The invention further provides for the use of identified therapeutic in treatment and/or prevention of osteoporosis. Finally, the invention provides a method for detg. the effectiveness of therapeutic in a subject who has or is predisposed to developing osteoporosis.

L33 ANSWER 2 OF 3 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2000:437912 BIOSIS
DOCUMENT NUMBER: PREV200000437912
TITLE: **IL-1** receptor antagonist gene polymorphism associated with age of **menopause**.
AUTHOR(S): **van Dijk, S. (1)**; Stone, K. L.; Hannon, R. A.; Lui, L. L.; Sorrell, J. A.; Eastell, R.; Cummings, S. R.; **Duff, G. W.**
CORPORATE SOURCE: (1) Interleukin Genetics, Inc., San Antonio, TX USA
SOURCE: Journal of Bone and Mineral Research, (September, 2000) Vol. 15, No. Suppl. 1, pp. S537. print. Meeting Info.: Twenty-Second Annual Meeting of the American Society for Bone and Mineral Research Toronto, Ontario, Canada September 22-26, 2000 American Society for Bone and Mineral Research . ISSN: 0884-0431.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English

L33 ANSWER 3 OF 3 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 2000:886548 SCISEARCH
THE GENUINE ARTICLE: 346YJ
TITLE: **IL-1** receptor antagonist gene polymorphism associated with age of **menopause**.
AUTHOR: vanDijk S (Reprint); Stone K L; Hannon R A; Lui L L; Sorrell J A; Eastell R; Cummings S R; **Duff G W**
CORPORATE SOURCE: INTERLEUKIN GENET INC, SAN ANTONIO, TX; UNIV CALIF SAN FRANCISCO, SAN FRANCISCO, CA 94143; UNIV SHEFFIELD, SHEFFIELD, S YORKSHIRE, ENGLAND
COUNTRY OF AUTHOR: USA; ENGLAND
SOURCE: JOURNAL OF BONE AND MINERAL RESEARCH, (SEP 2000) Vol. 15, Supp. [1], pp. M341-M341. Publisher: AMER SOC BONE & MINERAL RES, 2025 M ST, N W, STE 800, WASHINGTON, DC 20036-3309. ISSN: 0884-0431.
DOCUMENT TYPE: Conference; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 0

L34 41 S VANDIJK S?/AU

L35 1 S L34 AND L20
L36 0 S L35 NOT L32

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Searched: 1797656 seqs, 10463268293 residues

Total number of hits satisfying chosen parameters: 3595312

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Post-processing: Minimum Match 0%
Maximum Match 100%
Listing first 45 summaries

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30: em_htg_hum:*
31: em_htg_inv:*
32: em_htg_other:*
33: em_htgo_inv:*

Pred. No. is the number of results predicted by chance to have a score greater than or equal to the score of the result being printed, and is derived by analysis of the total score distribution.

SUMMARIES

Result No.	Score	% Match	Length	DB	ID	Description
1	20	100.0	20	6	AX015634	AX015634 Sequence
2	20	100.0	20	6	AX052810	AX052810 Sequence
3	20	100.0	20	6	AX067270	AX067270 Sequence
4	20	100.0	213	9	HS087XA1	Z16545 H. sapiens
5	20	100.0	125685	2	AC069268	AC069268 Homo sapi
c 6	20	100.0	142432	5	AC017074	AC017074 Homo sapi
7	20	100.0	145465	2	AC069399	AC069399 Homo sapi
c 8	20	100.0	185276	2	AC104653	AC104653 Homo sapi
c 9	19	95.0	147777	2	AL627261	Danio rer
c 10	18.4	92.0	66207	2	AC026534	AC026534 Homo sapi
11	18.4	92.0	153503	2	AC079836	AC079836 Homo sapi
12	18.4	92.0	167540	2	AF002473	AF002473 Homo sapi
13	18.4	92.0	170481	2	AC009108	AC009108 Homo sapi
14	18.4	92.0	171707	2	AC091151	AC091151 Homo sapi
15	18.4	92.0	195848	2	AC007989	AC007989 Homo sapi
c 16	18.4	92.0	204050	1	AJ414154	AJ414154 Yersinia
c 17	17.4	87.0	112424	5	AC092838	AC092838 Homo sapi
c 18	17.4	87.0	115905	2	AC096477	AC096477 Rattus no
19	17.4	87.0	117927	5	AC013416	AC013416 Homo sapi
20	17.4	87.0	130588	5	HS364I22	AL031012 Human DNA
21	17.4	87.0	152880	2	AC063919	AC063919 Homo sapi
c 22	17.4	87.0	180104	9	DJ526N18	AF123462 Homo sapi
c 23	17.4	87.0	190948	2	AC091342	AC091342 Rattus no
24	17.4	87.0	215586	2	AC073435	AC073435 Homo sapi
c 25	17	85.0	5612	1	AF043470	AF043470 Escherich
26	17	85.0	7395	1	EC74P0157	Y11275 E.coli 7 4
27	17	85.0	7395	6	AX191728	AX191728 Sequence
c 28	17	85.0	61505	9	AL512330	AL512330 Human DNA
29	17	85.0	92077	1	AF074613	AF074613 Escherich
30	17	85.0	92077	6	AX191727	AX191727 Sequence
31	17	85.0	92721	1	AB011549	AB011549 Escherich
32	17	85.0	92721	6	AX191725	AX191725 Sequence
33	17	85.0	113920	9	AL512284	AL512284 Human DNA
c 34	17	85.0	158602	9	AL133283	AL133283 Human DNA
35	17	85.0	175149	2	AL391218	AL391218 Homo sapi
c 36	17	85.0	175583	9	AL590502	AL590502 Human DNA
37	17	85.0	315761	2	AL158079	AL158079 Homo sapi
38	16.8	84.0	1458	6	PRAALAD	M71235 Pisum sativ
39	16.8	84.0	2053	1	ECOHSC	U05338 Escherichia
40	16.8	84.0	3768	1	ECOHSCA	U01827 Escherichia
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c 42	16.8	84.0	11598	1	AE005482	AE005482 Escherich
c 43	16.8	84.0	13332	1	D90883	D90883 E.coli geno
c 44	16.8	84.0	16238	1	D90882	D90882 E.coli geno
c 45	16.8	84.0	28067	3	CELF21E9	AF016663 Caenorhat

ALIGNMENTS

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Result    1
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LOCUS       AX015634                 20 bp    DNA     linear   PAT 07-SEP-2000
DEFINITION  Sequence 9 from Patent WO9950451.
ACCESSION   AX015634
VERSION     AX015634.1  GI:10041469
KEYWORDS    .
SOURCE      synthetic construct.
  ORGANISM  synthetic construct
            artificial sequence
REFERENCE   1  (bases 1 to 20)
  AUTHORS   Bhattacharyya,S., Leaves,N., Cookson,W.O. and Moffatt,M.F.
  TITLE     Polymorphism 1: linkage of asthma to a locus on chromosome 2
  JOURNAL   Patent: WO 9950451-A 9 07-OCT-1999;
            BHATTACHARYYA SUMIT (GB); ISIS INNOVATION (GB); LEAVES NICHOLAS
            (GB); COOKSON WILLIAM OSMOND CHARLES (GB); MOFFATT MIRIAM FLEUR
            (GB)
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            Bao,W., Sun,Y., Wu,Q., Wang,H., Yang,X., Cheng,C., Wang,Y., Xie,Y.
            Qi,X., Li,T., Zhang,H., Liu,N., Wu,D., Yu,S., Fan,H., Liu,Y.,
            Li,G., Li,C., Bao,Q., Bao,J., Wang,X., Song,L., Zhang,L., Guo,D.,
            Huang,F., Zhang,G., Li,J., Bian,X., Zhang,M., Li,L., Feng,X., Yu,J.
            and Yang,H.
  TITLE     Chromosome 3p genomic sequence
  JOURNAL   Unpublished
REFERENCE   2  (bases 1 to 125685)
  AUTHORS   Wang,J., Hu,S., Dong,W., Wang,J., Zhang,Y., Zhang,H., Liu,B.,
            Bao,W., Sun,Y., Wu,Q., Wang,H., Yang,X., Cheng,C., Wang,Y., Xie,Y.,
            Qi,X., Li,T., Zhang,H., Liu,N., Wu,D., Yu,S., Fan,H., Liu,Y.,
            Li,G., Li,C., Bao,Q., Bao,J., Wang,X., Song,L., Zhang,L., Guo,D.,
            Huang,F., Zhang,G., Li,J., Bian,X., Zhang,M., Li,L., Feng,X., Yu,J.
            and Yang,H.
  TITLE     Direct Submission
  JOURNAL   Submitted (23-MAY-2000) Human Genomic Center, Institute of
            Genetics, Chinese Academy of Sciences, Datun Road, Beijing, Beijing
            100101, P.R.China
COMMENT     Center code: Beijing.
            * NOTE: This is a 'working draft' sequence. It currently
            * consists of 31 contigs. The true order of the pieces
            * is not known and their order in this sequence record is
            * arbitrary. Gaps between the contigs are represented as
            * runs of N, but the exact sizes of the gaps are unknown.
            * This record will be updated with the finished sequence
            * as soon as it is available and the accession number will
            * be preserved.
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            *  10314     12296: contig of 1983 bp in length
            *                   gap of unknown length
            *  12297     15201: contig of 2905 bp in length
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            *  15202     17332: contig of 2131 bp in length
            *                   gap of unknown length
            *  17333     19575: contig of 2243 bp in length
            *                   gap of unknown length
            *  19576     22825: contig of 3250 bp in length
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            *  22826     25466: contig of 2641 bp in length
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            *  61175     65975: contig of 4801 bp in length
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DEFINITION  Homo sapiens BAC clone RP11-432G15 from 2, complete sequence.
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VERSION     AC017074.9  GI:18093142
KEYWORDS    HTG.
SOURCE      human.
  ORGANISM  Homo sapiens
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            Mammalia; Eutheria; Primates; Catarrhini; Hominidae; Homo.
REFERENCE   1  (bases 1 to 142432)
  AUTHORS   Sulston,J.E. and Waterston,R.
  TITLE     Toward a complete human genome sequence
  JOURNAL   Genome Res. 8 (11), 1097-1108 (1998)
  MEDLINE   99063792
REFERENCE   2  (bases 1 to 142432)
  AUTHORS   Fourman,C., Haakenson,W., Dignam,C. and Dixon,R.
  TITLE     The sequence of Homo sapiens BAC clone RP11-432G15
  JOURNAL   Unpublished (2001)
REFERENCE   3  (bases 1 to 142432)
  AUTHORS   Waterston,R.H.
  TITLE     Direct Submission
  JOURNAL   Submitted (09-DEC-1999) Genome Sequencing Center, Washington
            University School of Medicine, 4444 Forest Park Parkway, St. Louis,
            MO 63108, USA
REFERENCE   4  (bases 1 to 142432)
  AUTHORS   Waterston,R.
  TITLE     Direct Submission
  JOURNAL   Submitted (09-JAN-2002) Department of Genetics, Washington
            University, 4444 Forest Park Avenue, St. Louis, Missouri 63108, USA
COMMENT     On Jan 9, 2002 this sequence version replaced gi:14349336.
            ---------- Genome Center
                Center: Washington University Genome Sequencing Center
                Center code: WUGSC
                Web site: http://genome.wustl.edu/gsc
                Contact: sapiens@watson.wustl.edu
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                Center project name: H_NH0432G15
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RESULT   7
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DEFINITION  Homo sapiens chromosome 3 clone RP11-348I20 map 3p, WORKING DRAFT
            SEQUENCE, 46 unordered pieces.
ACCESSION   AC069399
VERSION     AC069399.2  GI:10312237
KEYWORDS    HTG; HTGS_PHASE1; HTGS_DRAFT.
SOURCE      human.
  ORGANISM  Homo sapiens
            Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
            Mammalia; Eutheria; Primates; Catarrhini; Hominidae; Homo
REFERENCE   1  (bases 1 to 145465)
  AUTHORS   Bao,J., Bao,Q., Bao,W., Bian,X., Cao,T., Chen,C., Chen,J., Ding,H.,
            Dong,W., Fan,H., Feng,X., Guan,Q., Gu,X., Guo,D., He,L., Hu,S.,
            Huang,F., Jin,Y., Kang,N., Li,C., Li,G., Li,J., Li,L.,
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            Luo,J., Niu,Y., Qi,Q., Qi,X., Song,S., Sun,N., Sun,W., Sun,Y.,
            Tao,R., Wang,H., Wang,J., Wang,L., Wang,L., Wang,R.,
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            Zhang,L., Zhang,M., Zhang,X., Zhang,X., Zhang,Y., Zhang,Y.,
            Zhang,Z., Zhu,B., Yu,J. and Yang,H.
  TITLE     Chromosome 3p genomic sequence
  JOURNAL   Unpublished
REFERENCE   2  (bases 1 to 145465)
  AUTHORS   Bao,J., Bao,Q., Bao,W., Bian,X., Cao,T., Chen,C., Chen,J., Ding,H.,
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            Zhang,L., Zhang,M., Zhang,X., Zhang,X., Zhang,Y., Zhang,Y.,
            Zhang,Z., Zhu,B., Yu,J. and Yang,H.
  TITLE     Direct Submission
  JOURNAL   Submitted (29-MAY-2000) Human Genomic Center, Institute Of
            Genetics, Chinese Academy of Sciences, Datun Road, Beijing, Beijing
            100101, P.R.China
COMMENT     On Sep 27, 2000 this sequence version replaced gi:8101198.
            ----------Genome Center
            Center:Beijing Center
            Center code:Beijing
            Website:http://hgc.igtp.ac.cn
            http://www.genomics.org.cn

Contact:hgc@igtp.ac.cn
-------------- Project Information
Center project name:bjx project
Center clone name: RP11-348I20
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Sequencing vector: puc18; 100% of reads
Chemistry: Dye-terminator; 55% of reads
Chemistry: Dye-terminator Big Dye; 45% of reads
Assembly program: Phrap; version 0.990329
Consensus quality: 117063 bases at least Q40
Consensus quality: 131502 bases at least Q30
Consensus quality: 142108 bases at least Q20
Insert size: 109473; sum-of-contigs
Quality coverage    3.15x in Q20 bases;sum-of-contigs
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* NOTE: This is a 'working draft' sequence. It currently
* consists of 46 contigs. The true order of the pieces
* is not known and their order in this sequence record is
* arbitrary. Gaps between the contigs are represented as
* runs of N, but the exact sizes of the gaps are unknown.
* This record will be updated with the finished sequence
* as soon as it is available and the accession number will
* be preserved.
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*     9091 10431: contig of 1341 bp in length
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     misc_feature    83113..89398
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     misc_feature    130524..140609
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     misc_feature    140710..161114
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                     /note="assembly_name:Contig59"
BASE COUNT    55292 a    37754 c    36132 g    52691 t     3407 others
ORIGIN
```

```
Smit, A.F.A. & Green, P. (1996-1997)
http://ftp.genome.washington.edu/RM/RepeatMasker.html
---------- Genome Center
    Center: Whitehead Institute/ MIT Center for Genome Research
    Center code: WIBR
    Web site: http://www-seq.wi.mit.edu
    Contact: sequence_submissions@genome.wi.mit.edu
---------- Project Information
    Center project name: L7064
    Center clone name: 362_L_21
----------.
* NOTE: This record contains 86 individual
* sequencing reads that have not been assembled into
* contigs. Runs of N are used to separate the reads
* and the order in which they appear is completely
* arbitrary. Low-pass sequence sampling is useful for
* identifying clones that may be gene-rich and allows
* overlap relationships among clones to be deduced.
* However, it should not be assumed that this clone
* will be sequenced to completion. In the event that
* the record is updated, the accession number will
* be preserved.
*       1      651: contig of 651 bp in length
*     652    751: gap of      100 bp
*     752   1406: contig of 655 bp in length
*    1407   1506: gap of      100 bp
*    1507   2175: contig of 669 bp in length
*    2176   2275: gap of      100 bp
*    2276   2970: contig of 695 bp in length
*    2971   3070: gap of      100 bp
*    3071   3729: contig of 659 bp in length
*    3730   3829: gap of      100 bp
*    3830   4461: contig of 632 bp in length
*    4462   4561: gap of      100 bp
*    4562   5221: contig of 660 bp in length
*    5222   5321: gap of      100 bp
*    5322   5984: contig of 663 bp in length
*    5985   6084: gap of      100 bp
*    6085   6755: contig of 671 bp in length
*    6756   6855: gap of      100 bp
*    6856   7542: contig of 687 bp in length
*    7543   7642: gap of      100 bp
*    7643   8303: contig of 661 bp in length
*    8304   8403: gap of      100 bp
*    8404   9073: contig of 670 bp in length
*    9074   9173: gap of      100 bp
*    9174   9825: contig of 652 bp in length
*    9826   9925: gap of      100 bp
*    9926  10641: contig of 716 bp in length
*   10642  10741: gap of      100 bp
*   10742  11434: contig of 693 bp in length
*   11435  11534: gap of      100 bp
*   11535  12216: contig of 682 bp in length
*   12217  12316: gap of      100 bp
*   12317  12967: contig of 651 bp in length
*   12968  13067: gap of      100 bp
*   13068  13716: contig of 649 bp in length
*   13717  13816: gap of      100 bp
*   13817  14487: contig of 671 bp in length
*   14488  14587: gap of      100 bp
*   14588  15250: contig of 663 bp in length
*   15251  15350: gap of      100 bp
*   15351  16062: contig of 712 bp in length
*   16063  16162: gap of      100 bp
*   16163  16839: contig of 677 bp in length
*   16840  16939: gap of      100 bp
*   16940  17611: contig of 672 bp in length
*   17612  17711: gap of      100 bp
*   17712  18352: contig of 641 bp in length
*   18353  18452: gap of      100 bp
*   18453  19151: contig of 699 bp in length
*   19152  19251: gap of      100 bp
*   19252  19958: contig of 707 bp in length
*   19959  20058: gap of      100 bp
*   20059  20741: contig of 683 bp in length
*   20742  20841: gap of      100 bp
*   20842  21495: contig of 654 bp in length
*   21496  21595: gap of      100 bp
*   21596  22222: contig of 627 bp in length
*   22223  22322: gap of      100 bp
*   22323  22991: contig of 669 bp in length
*   22992  23091: gap of      100 bp
*   23092  23756: contig of 665 bp in length
*   23757  23856: gap of      100 bp
*   23857  24524: contig of 668 bp in length
*   24525  24624: gap of      100 bp
*   24625  25293: contig of 669 bp in length
*   25294  25393: gap of      100 bp
*   25394  26103: contig of 710 bp in length
*   26104  26203: gap of      100 bp
*   26204  26849: contig of 646 bp in length
*   26850  26949: gap of      100 bp
*   26950  27627: contig of 678 bp in length
*   27628  27727: gap of      100 bp
*   27728  28388: contig of 661 bp in length
*   28389  28488: gap of      100 bp
*   28489  29152: contig of 664 bp in length
*   29153  29252: gap of      100 bp
*   29253  29918: contig of 666 bp in length
*   29919  30018: gap of      100 bp
*   30019  30684: contig of 666 bp in length
*   30685  30784: gap of      100 bp
*   30785  31451: contig of 667 bp in length
*   31452  31551: gap of      100 bp
*   31552  32202: contig of 651 bp in length
*   32203  32302: gap of      100 bp
*   32303  32985: contig of 683 bp in length
*   32986  33085: gap of      100 bp
*   33086  33811: contig of 726 bp in length
*   33812  33911: gap of      100 bp
*   33912  34600: contig of 689 bp in length
*   34601  34700: gap of      100 bp
*   34701  35384: contig of 684 bp in length
*   35385  35484: gap of      100 bp
*   35485  36164: contig of 680 bp in length
*   36165  36264: gap of      100 bp
*   36265  36994: contig of 730 bp in length
*   36995  37094: gap of      100 bp
*   37095  37845: contig of 751 bp in length
*   37846  37945: gap of      100 bp
*   37946  38600: contig of 655 bp in length
*   38601  38700: gap of      100 bp
*   38701  39355: contig of 655 bp in length
*   39356  39455: gap of      100 bp
*   39456  40104: contig of 649 bp in length
*   40105  40204: gap of      100 bp
*   40205  40847: contig of 643 bp in length
*   40848  40947: gap of      100 bp
*   40948  41595: contig of 648 bp in length
*   41596  41695: gap of      100 bp
*   41696  42369: contig of 674 bp in length
*   42370  42469: gap of      100 bp
*   42470  43191: contig of 722 bp in length
*   43192  43291: gap of      100 bp
*   43292  43972: contig of 681 bp in length
*   43973  44072: gap of      100 bp
*   44073  44729: contig of 657 bp in length
*   44730  44829: gap of      100 bp
*   44830  45500: contig of 671 bp in length
*   45501  45600: gap of      100 bp
*   45601  46248: contig of 648 bp in length
*   46249  46348: gap of      100 bp
*   46349  47009: contig of 661 bp in length
*   47010  47109: gap of      100 bp
*   47110  47773: contig of 664 bp in length
*   47774  47873: gap of      100 bp
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     misc_feature    25433..27718
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     misc_feature    27819..30797
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     misc_feature    30898..35000
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     misc_feature    65213..74226
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                     vector_side:right"
BASE COUNT    46008 a  31523 c  29971 g  43997 t   2404 others
ORIGIN

  Query Match             92.0%;  Score 18.4;  DB 2;  Length 153903;
  Best Local Similarity   95.0%;  Pred. No. 39;
  Matches   19;  Conservative    0;  Mismatches    1;  Indels    0;  Gaps    0;

Qy       1 gctgatattctggtgggaaa 20
           ||| ||||||||||||||||
Db   70911 GCTTATATTCTGGTGGGAAA 70930


RESULT 12
AP002473
LOCUS       AP002473               167540 bp    DNA     linear   HTG 08-JUN-2000
DEFINITION  Homo sapiens chromosome 18 clone RP11-745H17 map 18q12, WORKING
            DRAFT SEQUENCE, 22 unordered pieces.
ACCESSION   AP002473
VERSION     AP002473.1  GI:8388836
KEYWORDS    HTG; HTGS_PHASE1; HTGS_DRAFT.
SOURCE      Homo sapiens DNA, clone:RP11-745H17.
  ORGANISM  Homo sapiens
            Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
            Mammalia; Eutheria; Primates; Catarrhini; Hominidae; Homo.
REFERENCE   1  (bases 1 to 167540)
  AUTHORS   Hattori,M., Ishii,K., Toyoda,A., Taylor,T.D., Hong-Seog,P.,
            Fujiyama,A., Yada,T., Totoki,Y., Watanabe,H. and Sakaki,Y.
  TITLE     Homo sapiens 167,540 genomic DNA of 18q12
  JOURNAL   Published Only in DataBase (2000); In press
REFERENCE   2  (bases 1 to 167540)
  AUTHORS   Hattori,M., Ishii,K., Toyoda,A., Taylor,T.D., Hong-Seog,P.,
            Fujiyama,A., Yada,T., Totoki,Y., Watanabe,H. and Sakaki,Y.
  TITLE     Direct Submission
  JOURNAL   Submitted (06-JUN-2000) Masahira Hattori, The Institute of Physical
            and Chemical Research (RIKEN), Genomic Sciences Center (GSC),
            Kitasato Univ., 1-15-1 Kitasato, Sagamihara, Kanagawa 228-8555,
            Japan (E-mail:hattori@gsc.riken.go.jp,
            URL:http://hgp.gsc.riken.go.jp/, Tel:81-42-778-9223,
            Fax:81-42-778-9324)
COMMENT     ----------- Genome Center
            Center: RIKEN Genomic Sciences Center(GSC)
            Center code: RIKEN
            Web site: http://hgp.gsc.riken.go.jp/
            Contact: hattori@gsc.riken.go.jp
            ----------- Project Information
            Center project name: HumDraft18
            Center clone name: RP11-745H17
            ----------- Summary Statistics
            Sequencing vector: PCR products: 100% of reads
            Chemistry: Dye-terminator ET-amersham: 100% of reads
            Assembly program: Phrap; version 0.990329
            Consensus quality: 156364 bases at least Q40
            Consensus quality: 161470 bases at least Q30
            Consensus quality: 164021 bases at least Q20
            Insert size: 163410, sum-of-contigs
            Quality coverage: 5.37x in Q20 bases; sum-of-contigs
            -----------
            NOTE: This is a 'working draft' sequence. It currently consists of
            22 contigs. The true order of the pieces is not known and their
            order in this sequence record is arbitrary. Gaps between the
            contigs are represented as runs N, but the exact sizes of the gaps
            are unknown. This record will be updated with the finished sequence
            as soon as it is available and the accession number will be
            preserved.
                  1    26680 contig of  26680 bp in length
              26781    43880 contig of  17100 bp in length
              43881    61316 contig of  17436 bp in length
              61417    77415 contig of  15999 bp in length
              77516    90011 contig of  12496 bp in length
              90112   102577 contig of  12466 bp in length
             102678   112227 contig of   9550 bp in length
             112328   122049 contig of   9722 bp in length
             122150   126655 contig of   4506 bp in length
             126756   132018 contig of   5263 bp in length
             132119   136969 contig of   4851 bp in length
             137070   141509 contig of   4440 bp in length
             141610   147009 contig of   5400 bp in length
             147110   150145 contig of   3036 bp in length
             150246   153171 contig of   2926 bp in length
             153272   155871 contig of   2600 bp in length
             155972   156760 contig of   2789 bp in length
             156861   161580 contig of   2720 bp in length
             161681   163325 contig of   1645 bp in length
             163426   164786 contig of   1361 bp in length
             164887   166415 contig of   1529 bp in length
             166516   167540 contig of   1025 bp in length
            * NOTE: This is a 'working draft' sequence. It currently
            * consists of 22 contigs. The true order of the pieces
            * is not known and their order in this sequence record is
            * arbitrary. Gaps between the contigs are represented as
            * runs of N, but the exact sizes of the gaps are unknown.
            * This record will be updated with the finished sequence
            * as soon as it is available and the accession number will
            * be preserved.
            *       1    26680: contig of 26680 bp in length
            *   26681 26760: gap of      100 bp
            *   26781    43880: contig of 17100 bp in length
            *   43881 43980: gap of      100 bp
            *   43981    61316: contig of 17336 bp in length
            *   61317 61416: gap of      100 bp
            *   61417    77415: contig of 15999 bp in length
            *   77416 77515: gap of      100 bp
            *   77516    90011: contig of 12496 bp in length
            *   90012 90111: gap of      100 bp
            *   90112   102577: contig of 12466 bp in length
            *  102578 102677: gap of      100 bp
```

```
            *    37913    38012: gap of unknown length
            *    38013   114721: contig of 76709 bp in length
            *   114722   114821: gap of unknown length
            *   114822   170481: contig of 55660 bp in length.

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                     /organism="Homo sapiens"
                     /db_xref="taxon:9606"
                     /chromosome="16"
                     /clone="RP11-46J09"
                     /clone_lib="RPCI human BAC library 11"
BASE COUNT    41131 a   43143 c   42602 g   43302 t     303 others
ORIGIN


  Query Match             92.0%;  Score 18.4;  DB 2;  Length 170461;
  Best Local Similarity   95.0%;  Pred. No. 39;
  Matches   19;  Conservative    0;  Mismatches    1;  Indels    0;  Gaps    0;

Qy      1 gctgatattctgctgtgggaaa 20
          ||| ||||||||||||||||||
Db 116586 GCTCATATTCTGGTGGGAAA 116605


RESULT  14
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LOCUS       AC091151               171707 bp    DNA     linear   HTG 31-JUL-2001
DEFINITION  Homo sapiens chromosome 18 clone RP11-749H17 map 18, *** SEQUENCING
            IN PROGRESS ***, 2 ordered pieces.
ACCESSION   AC091151
VERSION     AC091151.4  GI:16506886
KEYWORDS    HTG; HTGS_PHASE2; HTGS_FULLTOP; HTGS_ACTIVEFIN
SOURCE      human.
  ORGANISM  Homo sapiens
            Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
            Mammalia; Eutheria; Primates; Catarrhini; Hominidae; Homo.
REFERENCE   1  (bases 1 to 171707)
  AUTHORS   Birren,B., Linton,L., Nusbaum,C. and Lander,E.
  TITLE     Homo sapiens chromosome 18, clone RP11-749H17
  JOURNAL   Unpublished
REFERENCE   2  (bases 1 to 171707)
  AUTHORS   Birren,B., Linton,L., Nusbaum,C., Lander,E., Allen,N., Anderson,S.,
            Barna,N., Bastien,V., Boguslavkiy,L., Boukhgalter,B., Brown,A.,
            Camarata,J., Campopiano,A., Chang,J., Choepel,Y., Colangelo,M.,
            Collins,S., Collymore,A., Cooke,P., DeArellano,K., Dewar,K.,
            Diaz,J.S., Dolan,M., Doyle,M., Ferreira,P., Fitzhugh,W., Gaye,D.,
            Galagan,J., Gardyna,S., Ginde,S., Goyette,M., Graham,L.,
            Grand-Pierre,N., Hagos,B., Hesford,A., Horton,L., Hulme,W.,
            Iliev,I., Johnson,R., Jones,C., Karatas,A., LaRocque,K.,
            Lamazares,R., Landers,T., Lehoczky,J., Levine,R., Liu,G.,
            MacLean,C., Macdonald,P., Marquis,N., Matthews,C., McCarthi,M.,
            McEwan,P., McKernan,K., McPheeters,R., Meldrim,J., Mehwald,L.,
            Mehta,R., Rieback,M., Riley,R., Risse,C., Roqov,P., Romon,J.,
            Rosetti,M., Roy,A., Santos,R., Schauer,S., Schuppack,K., Sekhan,S.,
            Severy,P., Sougnez,C., Spencer,B., Stange-Thomann,N.,
            Stojanovic,N., Strauss,N., Subramanian,A., Talamas,J., Tesfaye,S.,
            Theodore,J., Travers,M., Travis,N., Trigilio,J., Vassiliev,R.,
            Viel,R., Vo,A., Wilson,B., Wu,X., Wyman,D., Ye,W.J., Young,S.,
            Zainoun,J., Zembek,L., Zimmer,A. and Zody,M.
  TITLE     Direct Submission
  JOURNAL   Submitted (01-APR-2001) Whitehead Institute/MIT Center for Genome
            Research, 320 Charles Street, Cambridge, MA 02141, USA
COMMENT     On Oct 28, 2001 this sequence version replaced gi:15421950.
            All repeats were identified using RepeatMasker:
            Smit, A.F.A. & Green, P. (1996-1997)
            http://ftp.genome.washington.edu/RM/RepeatMasker.html
            ---------- Genome Center ----------
                Center: Whitehead Institute/ MIT Center for Genome Research
                Center code: WIBR
                Web site: http://www-seq.wi.mit.edu
                Contact: Sequence_submissions@genome.wi.mit.edu
            ---------- Project Information ----------
                Center project name: L13202
                Center clone name: 749_H_17
            ----------
            * NOTE: This is a "working draft" sequence.  It currently
            * consists of 2 contigs. Gaps between the contigs
            * are represented as runs of N. The order of the pieces
            * is believed to be correct as given, however the sizes
            * of the gaps between them are based on estimates that have
            * provided by the submittor.
            * This sequence will be replaced
            * by the finished sequence as soon as it is available and
            * the accession number will be preserved.
            *     1   154357: contig of 154357 bp in length
            *  154358 154457: gap of      100 bp
            *  154458   171707: contig of 17250 bp in length.
FEATURES             Location/Qualifiers
     source          1..171707
                     /organism="Homo sapiens"
                     /db_xref="taxon:9606"
                     /chromosome="18"
                     /map="18"
                     /clone="RP11-749H17"
                     /clone_lib="RPCI-11 Human Male BAC"
BASE COUNT    45096 a   36903 c   36956 g   48164 t    1586 others
ORIGIN


  Query Match             92.0%;  Score 18.4;  DB 2;  Length 171707;
  Best Local Similarity   95.0%;  Pred. No. 39;
  Matches   19;  Conservative    0;  Mismatches    1;  Indels    0;  Gaps    0;

Qy      1 gctgatattctgctgtgggaaa 20
          ||| ||||||||||||||||||
Db 112934 GCTCATATTCTGGTGGGAAA 112953


RESULT  15
AC007989
LOCUS       AC007989               195848 bp    DNA     linear   HTG 20-MAY-2001
DEFINITION  Homo sapiens chromosome 18 clone RP11-463D17 map 18, WORKING DRAFT
            SEQUENCE, 4 unordered pieces.
ACCESSION   AC007989
VERSION     AC007989.3  GI:14150910
KEYWORDS    HTG; HTGS_PHASE1; HTGS_DRAFT; HTGS_FULLTOP.
SOURCE      human.
  ORGANISM  Homo sapiens
            Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
            Mammalia; Eutheria; Primates; Catarrhini; Hominidae; Homo.
REFERENCE   1  (bases 1 to 195848)
  AUTHORS   Birren,B., Linton,L., Nusbaum,C. and Lander,E.
  TITLE     Homo sapiens chromosome 18, clone RP11-463D17
  JOURNAL   Unpublished
REFERENCE   2  (bases 1 to 195848)
  AUTHORS   Birren,B., Linton,L., Nusbaum,C., Lander,E., Allen,N., Anderson,M.,
            Baker J., Baldwin,J., Barna,N., Beckerly,R., Bent,Z., Brown,A.,
            Castle,A., Cerny,J., Colangelo,M., Collins,S., Collymore,A.,
            Cooke,P., DeArellano,K., Depayre,E., Devon,K., Dewar,K.,
            Doucette-Stamm,L., Doyle,M., Ferreira,P., Fitzhugh,W., Forrest,C.,
            Funke,R., Gaye,D., Galagan,J., Gardyna,S., Gilbert,D., Grant,G.,
            Hagos,B., Hesford,A., Horton,L., Howland,J.C., Jones,C., Kann,L.,
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            Peterson,K., Pollara,V., Riley,R., Roberts,C., Roy,A., Severy,P.,
            Stange-Thomann,N., Stojanovic,N., Stone,C., Subramanian,A.,
            Tesfaye,S., Torruella-Miller,I., Vassiliev,R., Vo,A., Wagner,A.,
            Wheeler,J., Wu,X., Wyman,D., Ye,W.J. and Zody,M.
  TITLE     Direct Submission
  JOURNAL   Submitted (08-JUL-1999) Whitehead Institute/MIT Center for Genome
            Research, 320 Charles Street, Cambridge, MA 02141, USA
```

OM nucleic - nucleic search, using sw model

Run on: July 18, 2002, 03:35:46 ; Search time 373.42 Seconds
(without alignments)
91.956 Million cell updates/sec

Title: US-09-632-657-7
Perfect score: 20
Sequence: 1 gctgatattctggtgggaaa 20

Scoring table: IDENTITY_NUC
Gapop 10.0 , Gapext 1.0

Searched: 1736436 seqs, 858457221 residues

Total number of hits satisfying chosen parameters: 3472872

Minimum DB seq length: 0
Maximum DB seq length: 2000000000

Post-processing: Minimum Match 0%
Maximum Match 100%
Listing first 45 summaries

Database : N_Geneseq_032802.*
1: /SIDS1/gcgdata/geneseq/geneseqn-embl/NA1980.DAT:*
2: /SIDS1/gcgdata/geneseq/geneseqn-embl/NA1981.DAT:*
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5: /SIDS1/gcgdata/geneseq/geneseqn-embl/NA1984.DAT:*
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10: /SIDS1/gcgdata/geneseq/geneseqn-embl/NA1989.DAT:*
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18: /SIDS1/gcgdata/geneseq/geneseqn-embl/NA1997.DAT:*
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20: /SIDS1/gcgdata/geneseq/geneseqn-embl/NA1999.DAT:*
21: /SIDS1/gcgdata/geneseq/geneseqn-embl/NA2000.DAT:*
22: /SIDS1/gcgdata/geneseq/geneseqn-embl/NA2001A.DAT:*
23: /SIDS1/gcgdata/geneseq/geneseqn-embl/NA2001B.DAT:*
24: /SIDS1/gcgdata/geneseq/geneseqn-embl/NA2002.DAT:*

Pred. No. is the number of results predicted by chance to have a score greater than or equal to the score of the result being printed, and is derived by analysis of the total score distribution.

SUMMARIES

Result No.	Score	% Query Match	Length	DB	ID	Description
1	20	100.0	20	20	AAZ28417	PCR primer D2S121
2	20	100.0	20	21	AAC63773	Human IL-1 gene 5'
3	20	100.0	20	21	AAA61927	Human IL-1 gene cl
4	20	100.0	20	21	AAZ37222	PCR primer for Int
5	20	100.0	20	22	AAF77857	Human IL-1 polymor
6	20	100.0	20	22	AAF27670	Primer #3. Uniden
7	20	100.0	20	22	AAC91438	Human IL-1A polymo
8	20	100.0	20	22	AAC89183	Human microsatelli
c 9	16.8	84.0	300	20	AAZ14604	Human gene express
10	16.8	84.0	486	23	AAS88820	DNA encoding novel
11	16.8	84.0	2347	23	AAS88827	DNA encoding novel
c 12	16.8	84.0	2370	23	AAS88575	DNA encoding novel
13	16.8	84.0	2370	23	AAS90850	DNA encoding novel
14	16.8	84.0	2469	23	AAS88821	DNA encoding novel
15	16.8	84.0	2829	23	AAS88825	DNA encoding novel
16	16.8	84.0	3256	23	AAS92977	DNA encoding novel
c 17	16.8	84.0	3261	23	AAS89027	DNA encoding novel
c 18	16.8	84.0	10611	22	AAS46259	DNA encoding novel
19	16.4	82.0	1380	21	AAA54037	Murine serotonin 5
20	16.4	82.0	1380	21	AAA54038	Murine serotonin 5
21	16.4	82.0	1380	21	AAA54039	Murine serotonin 5
22	16.4	82.0	1380	21	AAA54040	Murine serotonin 5
23	16.4	82.0	1380	21	AAA54041	Murine serotonin 5
24	16.4	82.0	1380	21	AAA54042	Murine serotonin 5
25	16.4	82.0	1848	15	AAQ73589	Fragment of the ra
26	16.4	82.0	2246	10	AAW89955	Serotonin 5HT1c re
27	16.4	82.0	2246	16	AAQ74064	Rat serotonin-1c c
28	16.4	82.0	2246	19	AAV48290	Rat 5-HT2C seroton
29	16.4	82.0	2246	19	AAV48291	Rat 5-HT2C 5312F m
30	16.4	82.0	2246	19	AAV48288	Rat 5-HT2C serotn
31	16.4	82.0	2246	19	AAV48289	Rat 5-HT2C 5312K m
32	16.4	82.0	2246	19	AAV48279	Rat 5-HT2C serotn
c 33	15.8	79.0	218	21	AAC24854	Human secreted pro
c 34	15.8	79.0	326	16	AAT22639	Human gene signatu
c 35	15.8	79.0	501	22	AAK11775	Human brain expres
c 36	15.8	79.0	501	22	AAI43374	Probe #12060 used
c 37	15.8	79.0	664	21	AAC95069	Cat flea hindgut a
38	15.8	79.0	721	23	AAS64362	Human cervical can
c 39	15.8	79.0	1108	22	AAH72838	Human cervical can
40	15.8	79.0	1113	23	AAS78343	DNA encoding novel
c 41	15.8	79.0	1278	24	AAD25144	Human NOV6 DNA enc
c 42	15.8	79.0	1348	20	AAX60809	Human secreted pro
c 43	15.8	79.0	1348	22	AAS59215	Human cDNA encodin
c 44	15.8	79.0	1348	24	ABA90884	Human polynucleoti
c 45	15.8	79.0	1516	23	AAS65181	DNA encoding novel

ALIGNMENTS

RESULT 1
AAZ28417
ID AAZ28417 standard; DNA; 20 BP.
XX
AC AAZ28417;
XX
DT 20-DEC-1999 (first entry)
XX
DE PCR primer D2S121 used to amplify the D2S121 microsatellite marker.
XX
KW PCR primer; microsatellite marker; diagnosis; asthma; predisposition;
KW chromosome 2; genetic polymorphism; D2S121; detect; sa.
XX
OS Synthetic
OS Homo sapiens.
XX
PN WO9950451-A1.
XX
PD 07-OCT-1999.
XX
PF 26-MAR-1999; 99WO-GB00968.
XX
PR 27-MAR-1998; 98GB-0006652.
XX
PA (ISIS-) ISIS INNOVATION LTD.
XX
PI Cookson WOCM, Moffatt MF, Bhattacharyya S, Leaves N;
XX
DR WPI; 1999-601341/51.
XX
PT Diagnosing asthma, or an asthmatic predisposition, from the presence of
PI specific alleles at a locus on chromosome 2 -

```
CC   coagulation, multiple organ failure, cardiovascular collapse, and death.
CC   It is therefore useful to identify those individuals who are at risk of
CC   developing an exaggerated systemic inflammatory response to septic
CC   stimuli, so that sepsis therapy (e.g. with IL-1 modulators) can be
CC   tailored to the individual's genetic profile. Sequences AAA61915-A61928
CC   represent PCR primers which may be used to amplify IL-1 gene cluster
CC   polymorphic loci. Sequences AAA61925-A61926 are primers used to amplify
CC   and type the centromere distal polymorphic microsatellite anchor marker
CC   AFM087xal which flanks the IL-1 gene cluster.
XX
SQ   Sequence 20 BP; 5 A; 2 C; 7 G; 6 T; 0 other;

  Query Match             100.0%;  Score 20;  DB 21;  Length 20;
  Best Local Similarity   100.0%;  Pred. No. 0.89;
  Matches   20;  Conservative    0;  Mismatches    0;  Indels    0;  Gaps    0;

Qy       1 gctgatattctggtgggaaa 20
           ||||||||||||||||||||
Db       1 gctgatattctggtgggaaa 20


RESULT   4
AAZ37222
ID   AAZ37222 standard; DNA; 20 BP.
XX
AC   AAZ37222;
XX
DT   28-JAN-2000  (first entry)
XX
DE   PCR primer for interleukin-1 genomic locus.
XX
KW   PCR primer; IL-1; IL-1A; IL-1B; TNFA; interleukin-1; foetus;
KW   adverse pregnancy outcome; allele detection; low birth weight; LBW;
KW   pre-term baby; premature baby; tumour necrosis factor-alpha; ss.
XX
OS   Synthetic.
OS   Homo sapiens.
XX
PN   WO9954707-A2.
XX
PD   28-OCT-1999.
XX
PF   21-APR-1999;  99WO-US08794.
XX
PR   21-APR-1998;  98US-0082487.
XX
PA   (MEDI-) MEDICAL SCI SYSTEMS INC.
XX
PI   Kuhlman KS,  Offenbacher S,  Duff GW;
XX
DR   WPI; 2000-013279/01.
XX
PT   Foetal testing for prediction of low birth weight by detecting an
PT   interleukin allele -
XX
PS   Disclosure; Page 33; 78pp; English.
XX
CC   This sequence is a PCR primer for the interleukin-1 genomic locus.
CC   The invention relates to a method for determining whether a foetus is
CC   predisposed to having an adverse pregnancy outcome by detecting an
CC   interleukin-1A (IL-1A) (+4845) allele 2, an IL-1 (-511) allele 1 or an
CC   allele in linkage disequilibrium with either of these alleles. The method
CC   comprises: (a) obtaining a nucleic acid sample from the subject; and
CC   (b) detecting an IL-1A (+4845) allele 2, an IL-1 (-511) allele 1 or an
CC   allele in linkage disequilibrium with either of these alleles. Where
CC   detection of the alleles or an allele in linkage disequilibrium with
CC   them indicates that the foetus is predisposed to an adverse pregnancy
CC   outcome. The method is used to determine whether a foetus is predisposed
CC   to having a low birth weight (LBW). By determining the IL-1 or tumour
CC   necrosis factor-alpha (TNF-A) genotype an appropriate therapeutic, such
CC   as a corticosteroid, antimetabolite, cytotoxic drug, colchicine or
CC   anti-cytokine, can be administered to compensate for the LBW causative
CC   functional mutation. An appropriate therapeutic can be used to treat a
CC   subject predisposed to having a LBW baby, especially a pre-term or
CC   premature baby.
XX
SQ   Sequence 20 BP; 5 A; 2 C; 7 G; 6 T; 0 other;

  Query Match             100.0%;  Score 20;  DB 21;  Length 20;
  Best Local Similarity   100.0%;  Pred. No. 0.89;
  Matches   20;  Conservative    0;  Mismatches    0;  Indels    0;  Gaps    0;

Qy       1 gctgatattctggtgggaaa 20
           ||||||||||||||||||||
Db       1 gctgatattctggtgggaaa 20


RESULT   5
AAF77857
ID   AAF77857 standard; DNA; 20 BP.
XX
AC   AAF77857;
XX
DT   30-MAY-2001  (first entry)
XX
DE   Human IL-1 polymorphic marker AFM087zal PCR primer #1.
XX
KW   Human; IL-1; PCR primer; osteoporosis; interleukin; haplotype pattern;
KW   polymorphic marker; microsatellite marker; AFM087zal; ss.
XX
OS   Homo sapiens
XX
PN   WO200116377-A2.
XX
PD   08-MAR-2001.
XX
PF   30-AUG-2000;  2000WO-US23844.
XX
PR   30-AUG-1999;  99US-0151460.
XX
PA   (INTE-) INTERLEUKIN GENETICS INC.
XX
PI   Van Dijk S,  Duff GW;
XX
DR   WPI; 2001-265896/27.
XX
PT   Determining whether a female is predisposed to the developing
PT   osteoporosis involves identifying interleukin-1 haplotype pattern or
PT   the female, useful for selecting appropriate osteoporosis therapy
XX
PS   Disclosure; Page 22; 69pp; English.
XX
CC   The present invention relates to a method for determining whether a
CC   female is predisposed to developing osteoporosis. The method involves
CC   identifying the patient's interleukin (IL)-1 haplotype pattern (HP).
CC   Presence of HP 1 or HP 2 indicates that the female is susceptible to
CC   larger bone loss and/or increased risk of fracture during the early
CC   menopausal years (HP 1) or post-menopause (HP 2). The human IL-1 locus
CC   genes are flanked by a distal polymorphic marker designated
CC   microsatellite anchor marker AFM087xal. The present sequence is a PCR
CC   primer for the AFM087xal marker, which was used in the present invention.
XX
SQ   Sequence 20 BP; 5 A; 2 C; 7 G; 6 T; 0 other;

  Query Match             100.0%;  Score 20;  DB 22;  Length 20;
  Best Local Similarity   100.0%;  Pred. No. 0.89;
  Matches   20;  Conservative    0;  Mismatches    0;  Indels    0;  Gaps    0;

Qy       1 gctgatattctggtgggaaa 20
           ||||||||||||||||||||
Db       1 gctgatattctggtgggaaa 20
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XX
PR   26-MAY-1999;   99US-0320395.
PR   01-NOV-1999;   99US-0431352.
XX
PA   (INJE-) INTERLEUKIN GENETICS INC.
XX
PI   Francis SE, Crossman DC, Duff GW, Kornman KS;
XX
DR   WPI; 2001-032066/04.
XX
PT   Diagnosing cardiovascular disease, or susceptibility, useful e.g. for
PT   selecting treatment, from presence of disease-associated allele,
PT   particularly in interleukin-1 genes -
XX
PS   Disclosure; Page 50; 121pp; English.
XX
CC   There is an association of patterns of alleles at four polymorphic loci
CC   in the interleukin-1 (IL-1) gene cluster with cardiovascular disorders.
CC   The present invention relates to a method for the diagnosis of a
CC   cardiovascular disorder (CVD), via detecting a CVD-associated allele in a
CC   patient sample. The method is used to select an appropriate therapy for
CC   CVD, particularly fragile plaque disorder, occlusive disorders or
CC   in-stent restenosis, or a condition associated with a mutation with
CC   linkage disequilibrium with a CVD-associated allele, or to identify risk
CC   factors for CVD, and to formulate a treatment designed to reduce the
CC   risk. The IL-1 gene cluster is found at the 2q13 region of chromosome 2.
CC   IL-1 locus genes are flanked by a centromere proximal polymorphic marker
CC   designated APN220xe3 at 127.4cM and a distal polymorphic marker
CC   designated APM087xa1 at 127.9cM. The present sequence is a PCR primer
CC   used in the present invention.
XX
SQ   Sequence 20 BP; 5 A; 2 C; 7 G; 6 T; 0 other;

  Query Match           100.0%;  Score 20;  DB 22;  Length 20;
  Best Local Similarity  100.0%;  Pred. No. 0.89;
  Matches   20;  Conservative    0;  Mismatches    0;  Indels    0;  Gaps    0;

Qy       1 gctgatattctggtgggaaa 20
           ||||||||||||||||||||
Db       1 gctgatattctggtgggaaa 20


RESULT   9
AAZ14604/c
ID   AAZ14604 standard; cDNA; 300 BP.
XX
AC   AAZ14604;
XX
DT   12-OCT-1999  (first entry)
XX
DE   Human gene expression product cDNA sequence SEQ ID NO:2073.
XX
KW   Human; gene; gene expression product; diagnosis; therapy; prote:
KW   detection; mapping; tissue typing; profiling; forensic; cancer,
KW   genetic analysis; colorectal cancer; breast cancer; lung cancer; ss
XX
OS   Homo sapiens.
XX
PN   WO9938972-A2.
XX
PD   05-AUG-1999.
XX
PF   28-JAN-1999;   99WO-US01619.
XX
PR   03-APR-1998;   98US-0080666.
PR   28-JAN-1998;   98US-0072910.
PR   24-FEB-1998;   98US-0075954.
PR   31-MAR-1998;   98US-0080114.
PR   03-APR-1998;   98US-0080515.
XX
PA   (CHIR ) CHIRON CORP.
PA   (HYSE-) HYSEQ INC.
XX
PI   Cikvenjakov R, Dickson M, Drmanac R, Drmanac S,
PI   Escobedo J, Garcia PD, Garcia V, Giese K, Innis MA,
PI   Jones WL, Kassam A, Kennedy SC, Kita D, Latat I,
PI   Lamson G, Leshkowitz D, Pot D, Randazzo F, Reinhard C,
PI   Stache-Crain B, Sudduth-Klinger J, Williams LT;
XX
DR   WPI; 1999-494052/41.
XX
PT   Novel human genes and their expression products which are
PT   differentially expressed in different cell types
XX
PS   Claim 1; Page 1129; 2479pp; English.
XX
CC   The present invention describes a library of human polynucleotides
CC   comprising the sequences given in AA212532 to AA217779. Also described is
CC   a method of detecting differentially expressed genes correlated with the
CC   cancerous state of a mammalian cell, comprising detecting at least one
CC   differentially expressed gene product in a test sample from a cell
CC   suspected of being cancerous, where the gene product is encoded by one
CC   of the 5248 polynucleotide sequences given in AA212532 to AA217779. The
CC   polynucleotides can be used as a source of primers and probes, which can
CC   be used for a variety or purpose, e.g. detection of expression levels,
CC   mapping, tissue typing or profiling, forensics, genetic analysis and
CC   detection of polymorphisms. Polypeptides encoded by the polynucleotides
CC   can be used for raising antibodies for experimental, diagnostic and
CC   therapeutic purposes. The polynucleotides may also be used to construct
CC   arrays for diagnostics (which may be used to determine function of an
CC   encoded protein), and to detect differences in expression levels between
CC   two cells (e.g. to identify abnormal or diseased tissue in a human, to
CC   identify a genetic predisposition or susceptibility to a disease such as
CC   cancer). The polynucleotides of the invention are especially used in the
CC   diagnosis, prognosis and management of colorectal cancer, breast cancer,
CC   and lung cancer. The polynucleotides can also be used to screen for
CC   peptide analogues and antagonists.
XX
SQ   Sequence 300 BP; 76 A; 75 C; 70 G; 79 T; 0 other;

  Query Match           84.0%;  Score 16.8;  DB 20;  Length 300;
  Best Local Similarity  90.0%;  Pred. No. 52;
  Matches   18;  Conservative    0;  Mismatches    2;  Indels    0;  Gaps    0;

Qy       1 gctgatattctggtgggaaa 20
           |||||||||| || |||||
Db     166 GCTGATATTCTAGTTGGAAA 147


RESULT   10
AAS88820
ID   AAS88820 standard; cDNA; 486 BP.
XX
AC   AAS88820;
XX
DT   13-FEB-2002  (first entry)
XX
DE   DNA encoding novel human diagnostic protein #24624.
XX
KW   Human; chromosome mapping; gene mapping; gene therapy; forensic;
KW   food supplement; medical imaging; diagnostic; genetic disorder; ss.
XX
OS   Homo sapiens.
XX
PN   WO200175067-A2.
XX
PD   11-OCT-2001.
XX
PF   30-MAR-2001; 2001WO-US08631.
XX
PR   31-MAR-2000; 2000US-0540217.
PR   23-AUG-2000; 2000US-0649167.
XX
PA   (HYSE-) HYSEQ INC.
```

```
XX
DE   WPI; 2001-639362/73.
DR   P-PSDB; AB024388.
XX
PT   New isolated polynucleotide and encoded polypeptides, useful in
PT   diagnostics, forensics, gene mapping, identification of mutations
PT   responsible for genetic disorders or other traits and to assess
PT   biodiversity -
XX
PS   Claim 1; SEQ ID No 24379; 103pp; English.
XX
CC   The invention relates to isolated polynucleotide (I) and
CC   polypeptide (II) sequences. (I) is useful as hybridisation probes,
CC   polymerase chain reaction (PCR) primers, oligomers, and for chromosome
CC   and gene mapping, and in recombinant production of (II). The
CC   polynucleotides are also used in diagnostics as expressed sequence tags
CC   for identifying expressed genes. (I) is useful in gene therapy techniques
CC   to restore normal activity of (II) or to treat disease states involving
CC   (II). (II) is useful for generating antibodies against it, detecting or
CC   quantitating a polypeptide in tissue, as molecular weight markers and as
CC   a food supplement. (II) and its binding partners are useful in medical
CC   imaging of sites expressing (II). (I) and (II) are useful for treating
CC   disorders involving aberrant protein expression or biological activity.
CC   The polypeptide and polynucleotide sequences have applications in
CC   diagnostics, forensics, gene mapping, identification of mutations
CC   responsible for genetic disorders or other traits to assess biodiversity
CC   and to produce other types of data and products dependent on DNA and
CC   amino acid sequences. AAS64197-AAS94564 represent novel human
CC   diagnostic coding sequences of the invention.
CC   Note: The sequence data for this patent did not appear in the printed
CC   specification, but was obtained in electronic format directly from WIPO
CC   at ftp.wipo.int/pub/published_pct_sequences.
XX
SQ   Sequence 2370 BP; 579 A; 735 C; 599 G; 457 T; 0 other;

  Query Match          84.0%;  Score 16.8;  DB 23;  Length 2370;
  Best Local Similarity   90.0%;  Pred. No. 68;
  Matches   18;  Conservative    0;  Mismatches    2;  Indels    0;  Gaps    0;

Qy       1 gctgatattctggtgggaaa 20
           || ||||||||||||||||||
Db     144 GCGGATATTCTGGTGGGTAA 125


RESULT  13
AAS90850
ID   AAS90850 standard; cDNA; 2370 BP.
XX
AC   AAS90850;
XX
DT   13-FEB-2002  (first entry)
XX
DE   DNA encoding novel human diagnostic protein #26654.
XX
KW   Human; chromosome mapping; gene mapping; gene therapy; forensic;
KW   food supplement; medical imaging; diagnostic; genetic disorder; ss.
XX
OS   Homo sapiens.
XX
PN   WO200175067-A2.
XX
PD   11-OCT-2001.
XX
PF   30-MAR-2001; 2001WO-US08631.
XX
PR   31-MAR-2000; 2000US-0540217.
PR   23-AUG-2000; 2000US-0649167.
XX
PA   (HYSE-) HYSEQ INC.
XX
PI   Drmanac RT, Liu C, Tang YT;
XX
DR   WPI; 2001-639362/73.
DR   P-PSDB; AB024388.
XX
PT   New isolated polynucleotide and encoded polypeptides, useful in
PT   diagnostics, forensics, gene mapping, identification of mutations
PT   responsible for genetic disorders or other traits and to assess
PT   biodiversity -
XX
PS   Claim 1; SEQ ID No 26654; 103pp; English.
XX
CC   The invention relates to isolated polynucleotide (I) and
CC   polypeptide (II) sequences. (I) is useful as hybridisation probes,
CC   polymerase chain reaction (PCR) primers, oligomers, and for chromosome
CC   and gene mapping, and in recombinant production of (II). The
CC   polynucleotides are also used in diagnostics as expressed sequence tags
CC   for identifying expressed genes. (I) is useful in gene therapy techniques
CC   to restore normal activity of (II) or to treat disease states involving
CC   (II). (II) is useful for generating antibodies against it, detecting or
CC   quantitating a polypeptide in tissue, as molecular weight markers and as
CC   a food supplement. (II) and its binding partners are useful in medical
CC   imaging of sites expressing (II). (I) and (II) are useful for treating
CC   disorders involving aberrant protein expression or biological activity.
CC   The polypeptide and polynucleotide sequences have applications in
CC   diagnostics, forensics, gene mapping, identification of mutations
CC   responsible for genetic disorders or other traits to assess biodiversity
CC   and to produce other types of data and products dependent on DNA and
CC   amino acid sequences. AAS64197-AAS94564 represent novel human
CC   diagnostic coding sequences of the invention.
CC   Note: The sequence data for this patent did not appear in the printed
CC   specification, but was obtained in electronic format directly from WIPO
CC   at ftp.wipo.int/pub/published_pct_sequences.
XX
SQ   Sequence 2370 BP; 457 A; 599 C; 735 G; 579 T; 0 other;

  Query Match          84.0%;  Score 16.8;  DB 23;  Length 2370;
  Best Local Similarity   90.0%;  Pred. No. 68;
  Matches   18;  Conservative    0;  Mismatches    2;  Indels    0;  Gaps    0;

Qy       1 gctgatattctggtgggaaa 20
           || ||||||||||||||||||
Db    2227 gcggatattctggtgggtaa 2246


RESULT  14
AAS88821
ID   AAS88821 standard; cDNA; 2469 BP.
XX
AC   AAS88821;
XX
DT   13-FEB-2002  (first entry)
XX
DE   DNA encoding novel human diagnostic protein #24625.
XX
KW   Human; chromosome mapping; gene mapping; gene therapy; forensic;
KW   food supplement; medical imaging; diagnostic; genetic disorder; ss.
XX
OS   Homo sapiens.
XX
PN   WO200175067-A2.
XX
PD   11-OCT-2001.
XX
PF   30-MAR-2001; 2001WO-US08631.
XX
PR   31-MAR-2000; 2000US-0540217.
PR   23-AUG-2000; 2000US-0649167.
XX
PA   (HYSE-) HYSEQ INC.
XX
PI   Drmanac RT, Liu C, Tang YT;
XX
DR   WPI; 2001-639362/73.
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GenCore version 4.5

OM nucleic - nucleic search, using sw model

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Total number of hits satisfying chosen parameters: 767066

Minimum DB seq length: 0
Maximum DB seq length: 2000000000

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Maximum Match 100%
Listing first 45 summaries

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Pred. No. is the number of results predicted by chance to have a score greater than or equal to the score of the result being printed, and is derived by analysis of the total score distribution.

SUMMARIES

Result No.	Score	% Query Match	Length	DB	ID	Description
1	20	100.0	20	4	US-09-345-217-6	Sequence 6, Appli
2	16.4	82.0	1848	1	US-08-313-553-10	Sequence 10, Appl
3	16.4	82.0	1848	3	US-08-767-993-10	Sequence 10, Appl
4	16.4	82.0	2246	4	US-09-032-742-3	Sequence 3, Appli
5	16.4	82.0	2246	4	US-09-032-742-21	Sequence 21, Appl
6	16.4	82.0	2246	4	US-09-032-742-22	Sequence 22, Appl
7	16.4	82.0	2246	4	US-09-032-742-24	Sequence 24, Appl
8	16.4	82.0	2246	4	US-09-032-742-25	Sequence 25, Appl
c 9	15.8	79.0	1246	3	US-09-005-180A-2	Sequence 2, Appli
10	15.2	76.0	711	4	US-08-998-416-500	Sequence 500, App
11	15.2	76.0	10409	3	US-08-772-440-33	Sequence 33, Appl
c 12	15.2	76.0	11933	4	US-09-470-618-13	Sequence 13, Appl
c 13	15.2	76.0	11933	4	US-09-364-862-13	Sequence 13, Appl
c 14	14.8	74.0	1313	1	US-08-461-731-3	Sequence 3, Appli
c 15	14.8	74.0	1313	2	US-09-002-072B-3	Sequence 3, Appli
c 16	14.8	74.0	1313	3	US-09-314-199-3	Sequence 3, Appli
c 17	14.8	74.0	1313	5	PCT-US94-10465-1	Sequence 1, Appli
18	14.8	74.0	1377	3	US-09-292-071-26	Sequence 26, Appl
19	14.8	74.0	1377	3	US-09-292-071-28	Sequence 28, Appl
20	14.8	74.0	1377	3	US-09-292-069A-26	Sequence 26, Appl
21	14.8	74.0	1377	3	US-09-292-069A-28	Sequence 28, Appl
22	14.8	74.0	1377	3	US-09-418-721-22	Sequence 22, Appl
23	14.8	74.0	2733	1	US-08-310-271-1	Sequence 1, Appli
24	14.8	74.0	2733	4	US-09-032-742-9	Sequence 9, Appli
c 25	14.8	74.0	3011	1	US-07-821-716-1	Sequence 1, Appli
c 26	14.4	72.0	1684	2	US-08-999-811-21	Sequence 21, Appl
27	14.4	72.0	2428	3	US-08-475-742-15	Sequence 15, Appl
28	14.4	72.0	2698	2	US-08-677-298-1	Sequence 1, Appli
29	14.4	72.0	51259	3	US-08-781-891-209	Sequence 209, App
c 30	14.2	71.0	102	1	US-08-036-555B-141	Sequence 141, App
c 31	14.2	71.0	102	1	US-08-036-555B-176	Sequence 176, App
c 32	14.2	71.0	102	1	US-08-469-569-141	Sequence 141, App
c 33	14.2	71.0	102	1	US-08-469-569-176	Sequence 176, App
c 34	14.2	71.0	102	1	US-08-249-322A-141	Sequence 141, App
c 35	14.2	71.0	102	1	US-08-249-322A-176	Sequence 176, App
c 36	14.2	71.0	102	1	US-08-469-526A-141	Sequence 141, App
c 37	14.2	71.0	102	1	US-08-469-526A-176	Sequence 176, App
c 38	14.2	71.0	102	2	US-08-734-591A-141	Sequence 141, App
c 39	14.2	71.0	102	2	US-08-734-591A-176	Sequence 176, App
c 40	14.2	71.0	102	2	US-08-469-660-141	Sequence 141, App
c 41	14.2	71.0	102	2	US-08-469-660-176	Sequence 176, App
c 42	14.2	71.0	102	3	US-08-341-018-23	Sequence 23, Appl
c 43	14.2	71.0	102	3	US-08-341-018-25	Sequence 25, Appl
c 44	14.2	71.0	102	3	US-08-470-335-141	Sequence 141, App
c 45	14.2	71.0	102	4	US-08-735-021-176	Sequence 176, App

ALIGNMENTS

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RESULT 1
US-09-345-217-6
; Sequence 6, Application US/09345217
; Patent No. 6265142
; GENERAL INFORMATION:
;  APPLICANT: DUFF, GORDON W.
;  APPLICANT: COX, ANGELA
;  APPLICANT: CAMP, NICOLA J.
;  APPLICANT: DIGIOVINE, FRANCESCO S.
;  TITLE OF INVENTION: DIAGNOSTICS AND THERAPEUTICS FOR DISEASES ASSOCIATED
;  TITLE OF INVENTION: WITH AN IL-1 INFLAMMATORY HAPLOTYPE
;  FILE REFERENCE: HSA-010.02
;  CURRENT APPLICATION NUMBER: US/09/345,217
;  CURRENT FILING DATE: 1999-06-30
;  EARLIER APPLICATION NUMBER: PCT/GB98/01481
;  EARLIER FILING DATE: 1998-05-21
;  EARLIER APPLICATION NUMBER: 9711040.7
;  EARLIER FILING DATE: 1997-05-29
;  NUMBER OF SEQ ID NOS: 32
;  SOFTWARE: PatentIn Ver. 2.0
; SEQ ID NO 6
;  LENGTH: 20
;  TYPE: DNA
;  ORGANISM: Artificial Sequence
;  FEATURE:
;  OTHER INFORMATION: Description of Artificial Sequence: primer
US-09-345-217-6

  Query Match             100.0%;  Score 20;  DB 4;  Length 20;
  Best Local Similarity   100.0%;  Pred. No. 0.098;
  Matches   20;  Conservative    0;  Mismatches    0;  Indels    0;  Gaps    0;

Qy      1 gctgatattctggtgggaaa 20
          ||||||||||||||||||||
Db      1 gctgatattctggtgggaaa 20


RESULT 2
US-08-313-553-10
; Sequence 10, Application US/08313553
; Patent No. 5641650
;  GENERAL INFORMATION:
;    APPLICANT:  TURNER, George J.
;    APPLICANT:  BEILACH, Mary C.
;    TITLE OF INVENTION:  EXPRESSION OF HETEROLOGOUS POLYPEPTIDES
;    TITLE OF INVENTION:  IN HALOBACTERIA
;    NUMBER OF SEQUENCES:  15
;    CORRESPONDENCE ADDRESS:
;      ADDRESSEE:  Walter H. Dreger
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;      CLASSIFICATION:  435
;    ATTORNEY/AGENT INFORMATION:
;      NAME:  Dreger, Walter H.
;      REGISTRATION NUMBER:  24,190
;      REFERENCE/DOCKET NUMBER:  A-57669/WHD
;    TELECOMMUNICATION INFORMATION:
;      TELEPHONE:  (415) 781-1989
;      TELEFAX:  (415) 398-3249
;  INFORMATION FOR SEQ ID NO:  10:
;    SEQUENCE CHARACTERISTICS:
;      LENGTH:  1848 base pairs
;      TYPE:  nucleic acid
;      STRANDEDNESS:  single
;      TOPOLOGY:  linear
;    MOLECULE TYPE:  cDNA
;    FEATURE:
;      NAME/KEY:  misc_feature
;      LOCATION:  376..414
;      OTHER INFORMATION:  /note= "Bacteriorhodopsin
;      OTHER INFORMATION:  pre-sequence."
;    FEATURE:
;      NAME/KEY:  terminator
;      LOCATION:  1756..1758
;      OTHER INFORMATION:  /note= "Bacteriorhodopsin stop
;      OTHER INFORMATION:  codon."
;    FEATURE:
;      NAME/KEY:  misc_feature
;      LOCATION:  517..591
;      OTHER INFORMATION:  /note= "Helix I of rat serotonin
;      OTHER INFORMATION:  receptor protein (Type 1C)."
;    FEATURE:
;      NAME/KEY:  misc_feature
;      LOCATION:  625..690
;      OTHER INFORMATION:  /note= "Helix II of rat serotonin
;      OTHER INFORMATION:  receptor protein (Type 1C)."
;    FEATURE:
;      NAME/KEY:  misc_feature
;      LOCATION:  736..807
;      OTHER INFORMATION:  /note= "Helix III of rat serotonin
;      OTHER INFORMATION:  receptor protein (Type 1C)."
;    FEATURE:
;      NAME/KEY:  misc_feature
;      LOCATION:  868..939
;      OTHER INFORMATION:  /note= "Helix IV of rat serotonin
;      OTHER INFORMATION:  receptor protein (Type 1C)."
;    FEATURE:
;      NAME/KEY:  misc_feature
;      LOCATION:  997..1059
;      OTHER INFORMATION:  /note= "Helix V of rat serotonin
;      OTHER INFORMATION:  receptor protein (Type 1C)."
;    FEATURE:
;      NAME/KEY:  misc_feature
;      LOCATION:  1297..1362
;      OTHER INFORMATION:  /note= "Helix VI of rat serotonin
;      OTHER INFORMATION:  receptor protein (Type 1C)."
;    FEATURE:
;      NAME/KEY:  misc_feature
;      LOCATION:  1411..1476
;      OTHER INFORMATION:  /note= "Helix VII of rat serotonin
;      OTHER INFORMATION:  receptor protein (Type 1C)."
;    FEATURE:
;      NAME/KEY:  mutation
;      LOCATION:  replace(213, "")
;      OTHER INFORMATION:  /note= "G to A mutation removes
;      OTHER INFORMATION:  AlwNI restriction site."
;    FEATURE:
;      NAME/KEY:  misc_feature
;      LOCATION:  1732..1734
;      OTHER INFORMATION:  /note= "Codon encoding the
;      OTHER INFORMATION:  C-terminal amino acid of the rat serotonin
;      OTHER INFORMATION:  receptor protein (Type 1C)."
;    FEATURE:
;      NAME/KEY:  misc_signal
;      LOCATION:  374
;      OTHER INFORMATION:  /note= "RNA start site."
;    FEATURE:
;      NAME/KEY:  CDS
;      LOCATION:  376..1734
;    FEATURE:
;      NAME/KEY:  terminator
;      LOCATION:  1735..1737
;      OTHER INFORMATION:  /note= "Serotonin stop codon."
;    FEATURE:
;      NAME/KEY:  repeat_region
;      LOCATION:  436..462
;      OTHER INFORMATION:  /note= "Sequence encoding
; Patent No. 6010885
;      OTHER INFORMATION:  polyaspartic acid."
;    FEATURE:
;      NAME/KEY:  mutation
;      LOCATION:  replace(1755, "")
;      OTHER INFORMATION:  /note= "C to T mutation removes
;      OTHER INFORMATION:  AlwNI restriction site."
US-08-767-993-10

  Query Match             82.0%;  Score 16.4;  DB 3;  Length 1848;
  Best Local Similarity   94.4%;  Pred. No. 13;
  Matches   17;  Conservative    0;  Mismatches    1;  Indels    0;  Gaps    0;

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Db     649 GCTGATATGCTGGTGGGA 666


RESULT  4
US-09-032-742-3
; Sequence 3, Application US/09032742
; Patent No. 6255089
;  GENERAL INFORMATION:
;    APPLICANT:  Teitler, Milt
;    APPLICANT:  Herrick-Davis, Katharine
;    APPLICANT:  Egan, Christina C.
;    TITLE OF INVENTION:  Constitutively Activated Serotonin
;    TITLE OF INVENTION:  Receptors
;    NUMBER OF SEQUENCES:  25
;    CORRESPONDENCE ADDRESS:
;      ADDRESSEE:  Laurence Weinberger
;      STREET:  882 S. Matlack Street, Suite 103
;      STREET:  P.O. Box 1663
;      CITY:  West Chester
;      STATE:  PA
;      COUNTRY:  USA
;      ZIP:  19380-0053
;    COMPUTER READABLE FORM:
;      MEDIUM TYPE:  Floppy disk
;      COMPUTER:  IBM PC compatible
;      OPERATING SYSTEM:  PC-DOS/MS-DOS
;      SOFTWARE:  PatentIn Release #1.0, Version #1.30
;    CURREN, APPLICATION DATA:
;      APPLICATION NUMBER:  US/09/032,742
;      FILING DATE:  27-FEB-1998
;      CLASSIFICATION:  536
;    ATTORNEY/AGENT INFORMATION:
;      NAME:  Weinberger, Laurence
;      REGISTRATION NUMBER:  27,965
;      REFERENCE/DOCKET NUMBER:  3086-4
;    TELECOMMUNICATION INFORMATION:
;      TELEPHONE:  (610) 431-1703
;      TELEFAX:  (610) 431-4181
;  INFORMATION FOR SEQ ID NO:  3:
;    SEQUENCE CHARACTERISTICS:
;      LENGTH:  2246 base pairs
;      TYPE:  nucleic acid
;      STRANDEDNESS:  single
;      TOPOLOGY:  linear
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;      APPLICATION NUMBER:  US/09/032,742
;      FILING DATE:  27-FEB-1998
;      CLASSIFICATION:  536
;    ATTORNEY/AGENT INFORMATION:
;      NAME:  Weinberger, Laurence
;      REGISTRATION NUMBER:  27,965
;      REFERENCE/DOCKET NUMBER:  3086-4
;    TELECOMMUNICATION INFORMATION:
;      TELEPHONE:  (610) 431-1703
;      TELEFAX:  (610) 431-4181
;  INFORMATION FOR SEQ ID NO:  24:
;    SEQUENCE CHARACTERISTICS:
;      LENGTH:  2246 base pairs
;      TYPE:  nucleic acid
;      STRANDEDNESS:  single
;      TOPOLOGY:  linear
;    MOLECULE TYPE:  DNA (genomic)
US-09-032-742-24

  Query Match             82.0%;  Score 16.4;  DB 4;  Length 2246;
  Best Local Similarity   94.4%;  Pred. No. 13;
  Matches   17;  Conservative    0;  Mismatches    1;  Indels    0;  Gaps    0;

Qy       1 gctgatatttctggtggga 18
           |||||||| |||||||||
Db     982 GCTGATATGCTGGTGGGA 999


RESULT   8
US-09-032-742-25
; Sequence 25, Application US/09032742
; Patent No. 6255089
;  GENERAL INFORMATION:
;    APPLICANT:  Teitler, Milt
;    APPLICANT:  Herrick-Davis, Katharine
;    APPLICANT:  Egan, Christina C.
;    TITLE OF INVENTION:  Constitutively Activated Serotonin
;    TITLE OF INVENTION:  Receptors
;    NUMBER OF SEQUENCES:  25
;    CORRESPONDENCE ADDRESS:
;      ADDRESSEE:  Laurence Weinberger
;      STREET:  882 S. Matlack Street, Suite 103
;      STREET:  P.O. Box 1663
;      CITY:  West Chester
;      STATE:  PA
;      COUNTRY:  USA
;      ZIP:  19380-0053
;    COMPUTER READABLE FORM:
;      MEDIUM TYPE:  Floppy disk
;      COMPUTER:  IBM PC compatible
;      OPERATING SYSTEM:  PC-DOS/MS-DOS
;      SOFTWARE:  PatentIn Release #1.0, Version #1.30
;    CURRENT APPLICATION DATA:
;      APPLICATION NUMBER:  US/09/032,742
;      FILING DATE:  27-FEB-1998
;      CLASSIFICATION:  536
;    ATTORNEY/AGENT INFORMATION:
;      NAME:  Weinberger, Laurence
;      REGISTRATION NUMBER:  27,965
;      REFERENCE/DOCKET NUMBER:  3086-4
;    TELECOMMUNICATION INFORMATION:
;      TELEPHONE:  (610) 431-1703
;      TELEFAX:  (610) 431-4181
;  INFORMATION FOR SEQ ID NO:  25:
;    SEQUENCE CHARACTERISTICS:
;      LENGTH:  2246 base pairs
;      TYPE:  nucleic acid
;      STRANDEDNESS:  single
;      TOPOLOGY:  linear
;    MOLECULE TYPE:  DNA (genomic)
US-09-032-742-25

  Query Match             82.0%;  Score 16.4;  DB 4;  Length 2246;
  Best Local Similarity   94.4%;  Pred. No. 13;
  Matches   17;  Conservative    0;  Mismatches    1;  Indels    0;  Gaps    0;

Qy       1 gctgatatttctggtggga 18
           |||||||| |||||||||
Db     982 GCTGATATGCTGGTGGGA 999


RESULT   9
US-09-005-180A-2/c
; Sequence 2, Application US/09005180A
; Patent No. 6124446
;  GENERAL INFORMATION:
;    APPLICANT:  Hillman, Jennifer L.
;    APPLICANT:  Corley, Neil C.
;    APPLICANT:  Shah, Purvi
;    TITLE OF INVENTION:  HUMAN VPS35/MEM3-RELATED PROTEIN
;    NUMBER OF SEQUENCES:  4
;    CORRESPONDENCE ADDRESS:
;      ADDRESSEE:  Incyte Pharmaceuticals, Inc.
;      STREET:  3174 Porter Dr.
;      CITY:  Palo Alto
;      STATE:  CA
;      COUNTRY:  USA
;      ZIP:  94304
;    COMPUTER READABLE FORM:
;      MEDIUM TYPE:  Diskette
;      COMPUTER:  IBM Compatible
;      OPERATING SYSTEM:  DOS
;      SOFTWARE:  FastSEQ for Windows Version 2.0
;    CURRENT APPLICATION DATA:
;      APPLICATION NUMBER:  US/09/005,180A
;      FILING DATE:  Filed January 8, 1998
;      CLASSIFICATION:  424
;    PRIOR APPLICATION DATA:
;      APPLICATION NUMBER:
;      FILING DATE:
;    ATTORNEY/AGENT INFORMATION:
;      NAME:  Billings, Lucy J.
;      REGISTRATION NUMBER:  36,749
;      REFERENCE/DOCKET NUMBER:  PF-0457 US
;    TELECOMMUNICATION INFORMATION:
;      TELEPHONE:  650-855-0555
;      TELEFAX:  650-845-4166
;      TELEX:
;  INFORMATION FOR SEQ ID NO:  2:
;    SEQUENCE CHARACTERISTICS:
;      LENGTH:  3246 base pairs
;      TYPE:  nucleic acid
;      STRANDEDNESS:  single
;      TOPOLOGY:  linear
;    IMMEDIATE SOURCE:
;      LIBRARY:  LUNGTUT06
;      CLONE:  2641812
US-09-005-180A-2

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  Best Local Similarity   89.5%;  Pred. No. 26;
  Matches   17;  Conservative    0;  Mismatches    2;  Indels    0;  Gaps    0;

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Db    1785 CTGATAGTCTGGTGGGCAA 1767


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US-08-998-416-900
; Sequence 900, Application US/08998416
; Patent No. 6239264
;  GENERAL INFORMATION:
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RESULT  12
US-09-470-618-13/c
; Sequence 13, Application US/09470618
; Patent No. 6200560
; GENERAL INFORMATION:
;  APPLICANT: Couto, Linda B.
;  APPLICANT: Colosi, Peter C.
;  TITLE OF INVENTION: Adeno-Associated Vectors for Expression of Factor VIII
;  TITLE OF INVENTION: by Target Cells
;  FILE REFERENCE: Avigen-04082
;  CURRENT APPLICATION NUMBER: US/09/470,618
;  CURRENT FILING DATE: 1999-12-22
;  EARLIER APPLICATION NUMBER: 09/364,862
;  EARLIER FILING DATE: 1999-07-30
;  EARLIER APPLICATION NUMBER: 60/125,974
;  EARLIER FILING DATE: 1999-03-24
;  EARLIER APPLICATION NUMBER: 60/104,994
;  EARLIER FILING DATE: 1998-10-20
;  NUMBER OF SEQ ID NOS: 15
;  SOFTWARE: PatentIn Ver. 2.0
; SEQ ID NO 13
;    LENGTH: 11933
;    TYPE: DNA
;    ORGANISM: Artificial Sequence
;    FEATURE:
;    OTHER INFORMATION: Description of Artificial Sequence:  Synthetic
US-09-470-618-13

  Query Match             76.0%;  Score 15.2;  DB 4;  Length 11933;
  Best Local Similarity   85.0%;  Pred. No. 70;
  Matches   17;  Conservative    0;  Mismatches    3;  Indels    0;  Gaps    0;

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Db    8451 GCTGGCATTCTGGAGGGAAA 8432


RESULT  13
US-09-364-862-13/c
; Sequence 13, Application US/09364862
; Patent No. 6221349
; GENERAL INFORMATION:
;  APPLICANT: Couto, Linda B.
;  APPLICANT: Colosi, Peter C.
;  TITLE OF INVENTION: ADENO-ASSOCIATED VECTORS FOR EXPRESSION OF FACTOR VIII
;  TITLE OF INVENTION: BY TARGET
;  TITLE OF INVENTION: CELLS
;  FILE REFERENCE: AVIGEN-03743
;  CURRENT APPLICATION NUMBER: US/09/364,862
;  CURRENT FILING DATE: 1999-07-30
;  EARLIER APPLICATION NUMBER: 60/125,974
;  EARLIER FILING DATE: 1999-03-24
;  EARLIER APPLICATION NUMBER: 60/104,994
;  EARLIER FILING DATE: 1998-10-20
;  NUMBER OF SEQ ID NOS: 14
;  SOFTWARE: PatentIn Ver. 2.0
; SEQ ID NO 13
;    LENGTH: 11933
;    TYPE: DNA
;    ORGANISM: Artificial Sequence
;    FEATURE:
;    OTHER INFORMATION: Description of Artificial Sequence:  synthetic
US-09-364-862-13

  Query Match             76.0%;  Score 15.2;  DB 4;  Length 11933;
  Best Local Similarity   85.0%;  Pred. No. 70;
  Matches   17;  Conservative    0;  Mismatches    3;  Indels    0;  Gaps    0;

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Db    8451 GCTGGCATTCTGGAGGGAAA 8432


RESULT  14
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; Sequence 3, Application US/08461731
; Patent No. 5716806
;  GENERAL INFORMATION:
;    APPLICANT:  MEISSNER, ET AL.
;    TITLE OF INVENTION:  Human Inositol Monophosphatase H1
;    NUMBER OF SEQUENCES:  9
;    CORRESPONDENCE ADDRESS:
;      ADDRESSEE:  CARELLA, BYRNE, BAIN, GILFILLAN,
;      ADDRESSEE:  CECCHI, STEWART & OLSTEIN
;      STREET:  6 BECKER FARM ROAD
;      CITY:  ROSELAND
;      STATE:  NEW JERSEY
;      COUNTRY:  USA
;      ZIP:  07068
;    COMPUTER READABLE FORM:
;      MEDIUM TYPE:  3.5 INCH DISKETTE
;      COMPUTER:  IBM PS/2
;      OPERATING SYSTEM:  MS-DOS
;      SOFTWARE:  WORD PERFECT 5.1
;    CURRENT APPLICATION DATA:
;      APPLICATION NUMBER:  US/08/461,731
;      FILING DATE:  June 5, 1995
;      CLASSIFICATION:  435
;    PRIOR APPLICATION DATA:
;      APPLICATION NUMBER:  PCT/US94/10465
;      FILING DATE:  16 SEP 94
;    ATTORNEY/AGENT INFORMATION:
;      NAME:  FERRARO, GREGORY D.
;      REGISTRATION NUMBER:  36,134
;      REFERENCE/DOCKET NUMBER:  325800-316
;    TELECOMMUNICATION INFORMATION:
;      TELEPHONE:  201-994-1700
;      TELEFAX:  201-994-1744
;  INFORMATION FOR SEQ ID NO:  3:
;    SEQUENCE CHARACTERISTICS:
;      LENGTH:  1313 BASE PAIRS
;      TYPE:  NUCLEIC ACID
;      STRANDEDNESS:  SINGLE
;      TOPOLOGY:  LINEAR
;    MOLECULE TYPE:  cDNA
US-08-461-731-3

  Query Match             74.0%;  Score 14.8;  DB 1;  Length 1313;
  Best Local Similarity   88.9%;  Pred. No. 80;
  Matches   16;  Conservative    0;  Mismatches    2;  Indels    0;  Gaps    0;

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Db    1202 TTATATTCTGGGGGAAA 1185


RESULT  15
US-09-002-072a-3/c
; Sequence 3, Application US/09002072B
; Patent No. 5955339
;  GENERAL INFORMATION:
;    APPLICANT:  Meissner, Paul et al.
;    TITLE OF INVENTION:  Human Inositol Monophosphatase H1
;    NUMBER OF SEQUENCES:  10
;    CORRESPONDENCE ADDRESS:
;      ADDRESSEE:  Human Genome Sciences, Inc.
;      STREET:  9410 Key West Avenue
;      CITY:  Rockville
;      STATE:  MD
;      COUNTRY:  USA
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GenCore version 4.5

OM nucleic - nucleic search, using sw model

Run on: July 18, 2002, 02:34:06 ; Search time 2539.01 Seconds
(without alignments)
106.317 Million cell updates/sec

Title: US-09-632-657-7
Perfect score: 20
Sequence: 1 gctgatattctggtgggaaa 20

Scoring table: IDENTITY_NUC
Gapop 10.0 , Gapext 1.0

Searched: 13736207 seqs, 6748477542 residues

Total number of hits satisfying chosen parameters: 27472414

Minimum DB seq length: 0
Maximum DB seq length: 2000000000

Post-processing: Minimum Match 0%
Maximum Match 100%
Listing first 45 summaries

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1: em_estba:*
2: em_esthum:*
3: em_estin:*
4: em_estmu:*
5: em_estov:*
6: em_estpl:*
7: em_estro:*
8: em_htc:*
9: gb_est1:*
10: gb_est2:*
11: gb_htc:*
12: gb_gss:*
13: em_gss_hum:*
14: em_gss_inv:*
15: em_gss_plo:*
16: em_gss_vrt:*

Pred. No. is the number of results predicted by chance to have a score greater than or equal to the score of the result being printed, and is derived by analysis of the total score distribution.

SUMMARIES

Result No.	Score	% Query Match	Length	DB	ID	Description
c 1	18	90.0	533	12	AQ410722	AQ410722 HS_5067_A
c 2	18	90.0	551	10	BM188182	BM188182 sa_z5f07
3	18	90.0	729	10	BE913740	BE913740 602f05z05
c 4	17.4	87.0	617	12	AZ458703	AZ458703 1M1_05105
c 5	17.4	87.0	651	10	BI066862	BI066862 pg31i_pk0
6	17.4	87.0	688	12	AG181383	AG181383 Pan trog1
7	17.4	87.0	697	12	AG181575	AG181575 Pan trog1
8	17	85.0	176	9	AV011849	AV011849 AV011849
9	17	85.0	195	9	AV293951	AV293951 AV293951
10	17	85.0	219	9	BB067695	BB067695 BB067695
11	17	85.0	248	9	BB047357	BB047357 BB047357
c 12	17	85.0	452	12	AQ819189	AQ819189 HS_5300_A
c 13	17	85.0	519	12	AQ437759	AQ437759 HS_5056_B
14	16.8	84.0	132	12	AQ420981	AQ420981 RPCI-11-2
c 15	16.8	84.0	154	10	BF758769	BF758769 MK3-C1045
c 16	16.8	84.0	218	9	AV333398	AV333398 AV333398
c 17	16.8	84.0	248	10	BF950269	BF950269 CM3-NN118
c 18	16.8	84.0	255	12	AQ267714	AQ267714 RPCI11-74
c 19	16.8	84.0	276	10	BF950329	BF950329 CM3-NN116
c 20	16.8	84.0	287	9	AI771884	AI771884 EST252984
21	16.8	84.0	335	9	AW521782	AW521782 OT-R-BOO-
22	16.8	84.0	345	10	BF186914	BF186914 EST443201
23	16.8	84.0	403	9	AW801704	AW801704 IL5-UM006
c 24	16.8	84.0	405	9	AI490092	AI490092 EST248431
c 25	16.8	84.0	415	9	AI897010	AI897010 EST266453
c 26	16.8	84.0	417	12	AQ014285	AQ014285 RPCI11-23
27	16.8	84.0	422	10	N21831	N21831 ZNSSN0015 r
28	16.8	84.0	427	10	BF188475	BF188475 EST444762
29	16.8	84.0	437	10	BF187039	BF187039 EST443326
30	16.8	84.0	451	9	AI484102	AI484102 EST249973
31	16.8	84.0	459	12	YA1H002Q	AL441749 T. brucei
32	16.8	84.0	484	10	BF187498	BF187498 EST443785
33	16.8	84.0	500	12	AQ420978	AQ420978 RPCI-11-2
34	16.8	84.0	558	12	AQ718936	AQ718936 HS_5513_B
35	16.8	84.0	570	12	AQ827212	AQ827212 HS_5297_A
36	16.8	84.0	577	12	AZ695565	AZ695565 RPCI-23-2
c 37	16.8	84.0	612	12	AZ274985	AZ274985 RPCI-23-1
38	16.8	84.0	621	10	BG350691	BG350691 096F02 MA
c 39	16.8	84.0	707	12	AQ162826	AQ162826 wgxb0016D
40	16.8	84.0	716	10	BG598861	BG598861 EST503761
41	16.8	84.0	764	10	BG889296	BG889296 EST515117
42	16.8	84.0	770	10	BI405164	BI405164 120G12 Ma
43	16.8	84.0	891	10	BF262165	BF262165 HV_CEa000
44	16.8	84.0	1021	12	CN506FKX	AL409495 T7 end of
45	16.8	84.0	1642	10	BI489140	BI489140 6030212135

ALIGNMENTS

```
RESULT 1
AQ410722/c
LOCUS       AQ410722                533 bp    DNA     linear   GSS 17-MAR-1999
DEFINITION  HS_5067_A1_C05_17A RPCI-11 Human Male BAC Library Homo sapiens
            genomic clone Plate=643 Col=5 Row=8, DNA sequence.
ACCESSION   AQ410722
VERSION     AQ410722.1  GI:4427915
KEYWORDS    GSS.
SOURCE      human.
  ORGANISM  Homo sapiens
            Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
            Mammalia; Eutheria; Primates; Catarrhini; Hominidae; Homo.
REFERENCE   1  (bases 1 to 533)
  AUTHORS   Mahairas,G.G., Wallace,J.C., Smith,K., Swartzell,S., Holzman,T.,
            Kelley,A., Shaker,R., Furlong,J., Young,J., Zhao,S., Adams,M.D. and
            Hood,L.
  TITLE     Sequence-tagged connectors: a sequence approach to mapping and
            scanning the human genome
  JOURNAL   Proc. Natl. Acad. Sci. U. S. A. 96 (17), 9739-9744 (1999)
  MEDLINE   99350589
COMMENT     Contact: Mahairas GG, Wallace JC, Hood L
            High Throughput Sequencing Center
            Univ. of Washington
            401 Queen Anne Avenue North, Seattle, WA 98109, USA
            Tel: (206) 616-3618
            Fax: (206) 616-3987
            Email: jwallace@u.washington.edu
            Clones are derived from the human BAC library RPCI-11. For BAC
            library availability, please contact Pieter de Jong
            (pdejong@med.buffalo.edu). Clones may be purchased from
            BACnAC Resources (http://bacpac.med.buffalo.edu/ordering_bac.htm)
            or from Research Genetics (info@resgen.com). BAC end Web Server:
            http://www.btac.washington.edu
            Plate: 643 row: E  column: 5
            Seq primer: T7
            Class: BAC ends
            High quality sequence stop: 533.
FEATURES    Location/Qualifiers
     source 1..533
            /organism="Homo sapiens"
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ORIGIN

  Query Match             90.0%;  Score 18;  DB 10;  Length 729;
  Best Local Similarity   100.0%;  Pred. No. 2.5e-02;
  Matches   18;  Conservative    0;  Mismatches    0;  Indels    0;  Gaps    0;

Qy       2 ctgatattctggtgggaa 19
           ||||||||||||||||||
Db     703 CTGATATTCTGGTGGGAA 720


RESULT   4
AZ458703/c
LOCUS       AZ458703                617 bp    DNA     linear   GSS 04-OCT-2000
DEFINITION  1M0263I05F Mouse 10kb plasmid UUGC1M library Mus musculus genomic
            clone UUGC1M0263I05 F, DNA sequence.
ACCESSION   AZ458703
VERSION     AZ458703.1  GI:10616828
KEYWORDS    GSS.
SOURCE      house mouse.
  ORGANISM  Mus musculus
            Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
            Mammalia; Eutheria; Rodentia; Sciurognathi; Muridae; Murinae; Mus.
REFERENCE   1  (bases 1 to 617)
  AUTHORS   Dunn,D., Aoyagi,A., Barber,M., Beacorn,T., Duval,B., Hamil,C.,
            Islam,H., Longacre,S., Mahmoud,M., Meenen,E., Pedersen,T., Reilly
            ,M., Rose,M., Rose,R., Stokes,R., Tingey,A., von Niederhausern,A.
            and Wright,D.,Weiss,R.
  TITLE     Mouse whole genome scaffolding with paired end reads from 10kb
            plasmid inserts
  JOURNAL   Unpublished (2000)
COMMENT     Contact: Robert B. Weiss
            University of Utah Genome Center
            University of Utah
            Rm. 30B, Biomedical Polymers Research Bldg., 20 S. 2030 E., SLC, UT
            84112, USA
            Tel: 801 585 5606
            Fax: 801 585 7177
            Email: ddunnegenetics.utah.edu
            Insert Length: 10000  Std Error: 0.00
            Plate: 0263  row: I  column: 05
            Seq primer: CGTTGTAAAACGACGGCCAGT
            Class: plasmid ends
            High quality sequence stop: 617.
FEATURES             Location/Qualifiers
     source          1..617
                     /organism="Mus musculus"
                     /strain="C57BL/6J"
                     /db_xref="taxon:10090"
                     /clone="UUGC1M0263I05"
                     /clone_lib="Mouse 10kb plasmid UUGC1M library"
                     /sex="Male"
                     /lab_host="E. Coli strain XL10-Gold, T1-resistant, F-"
                     /note="Vector: PWD42nv; Purified genomic DNA from M.
                     musculus C57BL/6J (male) was obtained  from the Jackson
                     Laboratory Mouse DNA Resource
                     (http://www.jax.org/resources/documents/dnares/). The DNA
                     was hydrodynamically sheared by repeated passage through a
                     0.005 inch orifice at constant velocity. The sheared DNA
                     was blunt end-repaired with T4 DNA polymerase and T4
                     polynucleotide kinase. Adaptor oligonucleotides were
                     ligated to the blunt ends in high  molar excess. The
                     adaptored DNA was purified and size-selected for a 9.5 to
                     10.5 kb range using preparative agarose gel
                     electrophoresis. Vector DNA was prepared from a derivative
                     of PWD42 (gi|4732114|gb|AF129072.1), a copy-number
                     inducible derivative of plasmid R1. The vector was ligated
                     with adaptors complementary to the insert adaptors and
                     purified. The sheared, adaptored mouse DNA was annealed to
                     adaptored vector DNA, and transformed into
                     chemically-competent E. coli XL10-Gold (Stratagene) cells
                     and selected for ampicillin resistance."
BASE COUNT      155 a    173 c    118 g    171 t
ORIGIN

  Query Match             87.0%;  Score 17.4;  DB 12;  Length 617;
  Best Local Similarity   94.7%;  Pred. No. 4.7e+02;
  Matches   18;  Conservative    0;  Mismatches    1;  Indels    0;  Gaps    0;

Qy       2 ctgatattctggtgggaa 20
           ||||| ||||||||||||
Db     396 CTGATGTTCTGGTGGGAAA 378


RESULT   5
B1066862/c
LOCUS       B1066862                651 bp    mRNA    linear   EST 15-JUN-2001
DEFINITION  pgfln.pk009.120 normalized chicken fat cDNA library Gallus gallus
            cDNA clone pgfln.pk009.120 5' similar to pir|JC4256|JC4256
            hypothetical 32.0k protein - Neurospora crassa gb|AAC41672.1|
            (L40806) open reading frame [Neurospora crassa] prf|2120297A ORF
            [Neurospora crassa]G, mRNA sequence
ACCESSION   B1066862
VERSION     B1066862.1  GI:14474384
KEYWORDS    EST.
SOURCE      chicken.
  ORGANISM  Gallus gallus
            Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
            Archosauria; Aves; Neognathae; Galliformes; Phasianidae;
            Phasianinae; Gallus.
REFERENCE   1  (bases 1 to 651)
  AUTHORS   Cogburn,L.A., Morgan,R.W. and Burnside,J.
  TITLE     Chicken EST8 from fat
  JOURNAL   Unpublished (2001)
COMMENT     Contact: Larry A. Cogburn
            University of Delaware
            Townsend Hall, Newark, DE 19717, USA
            Tel: 302-831-1335
            Fax: 302-831-2822
            Email: cogburn@udel.edu, www.chickest.udel.edu.
FEATURES             Location/Qualifiers
     source          1..651
                     /organism="Gallus gallus"
                     /db_xref="taxon:9031"
                     /clone="pgfln.pk009.120"
                     /clone_lib="normalized chicken fat cDNA library"
                     /sex="Male and Female"
                     /tissue_type="fat"
                     /lab_host="E.coli DH5H10B"
                     /note="Vector: pSPORT1"
BASE COUNT      174 a    141 c    166 g    158 t     12 others
ORIGIN

  Query Match             87.0%;  Score 17.4;  DB 10;  Length 651;
  Best Local Similarity   94.7%;  Pred. No. 4.8e+02;
  Matches   18;  Conservative    0;  Mismatches    1;  Indels    0;  Gaps    0;

Qy       1 gctgatattctggtgggaa 19
           |||||| ||||||||||||
Db     551 GCTGATGTTCTGGTGGGAA 533


RESULT   6
AG181383
LOCUS       AG181383                688 bp    DNA     linear   GSS 09-JAN-2002
DEFINITION  Pan troglodytes DNA, clone: RP43-054D23.17, genomic survey
            sequence.
ACCESSION   AG181383
VERSION     AG181383.1  GI:16711063
KEYWORDS    GSS; GSS (genome survey sequence).
SOURCE      Pan troglodytes male lymphocytes DNA, clone_lib:RPCI-43 Chimpanzee
            Male BAC Library clone:RP43-054D23.17.
  ORGANISM  Pan troglodytes
```

```
                     /clone_lib="Mus musculus 18-day embryo C57BL/6J"
                     /sex="mixed"
                     /dev_stage="18-day embryo"
BASE COUNT       40 a     41 c     43 g     52 t
ORIGIN


  Query Match             85.0%;  Score 17;  DB 9;  Length 176;
  Best Local Similarity   100.0%;  Pred. No. 5.9e+02;
  Matches   17;  Conservative    0;  Mismatches    0;  Indels    0;  Gaps    0;

Qy          2 ctgatattctggtggga 18
              |||||||||||||||||
Db         36 CTGATATTCTGGTGGGA 52


RESULT  9
AV293951
LOCUS       AV293951                 195 bp    mRNA    linear   EST 10-NOV-1999
DEFINITION  AV293951 RIKEN full-length enriched, 10 days neonate head Mus
            musculus cDNA clone 5530401C20 3', mRNA sequence.
ACCESSION   AV293951
VERSION     AV293951.1  GI:6325968
KEYWORDS    EST.
SOURCE      house mouse.
  ORGANISM  Mus musculus
            Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
            Mammalia; Eutheria; Rodentia; Sciurognathi; Muridae; Murinae; Mus.
REFERENCE   1  (bases 1 to 195)
  AUTHORS   Konno,H., Aizawa,K., Akahira,S., Akiyama,J., Carninci,P.,
            Fukuda,S., Fukunishi,Y., Hara,A., Hayatsu,N., Hirozane,T., Hori,F.,
            Ishii,Y., Ishikawa,T., Itoh,M., Izawa,M., Kadota,K., Kagawa,I., Kai
            ,C., Kawai,J., Kikuchi,N., Kojima,Y., Koya,S., Kusakabe,M.,
            Matsuyama,T., Miki,R., Mizuno,Y., Nakamura,M., Oda,H., Okazaki,Y.,
            Owa,C., Ozawa,Y., Saito,H., Sano,M., Sato,K., Shibata,K., Shibata
            ,Y., Shigemoto,Y., Shiraki,T., Sogabe,Y., Sugahara,Y., Suzuki,H.,
            Suzuki,H., Takahashi,F., Tateno,M., Tominaga,N., Tsunoda,Y.,
            Watahiki,A., Watanabe,S., Yamamura,T., Yasunishi,A., Yokota,T.,
            Yoshiki,A., Yoshino,M., Muramatsu,M. and Hayashizaki,Y.
  TITLE     RIKEN Mouse ESTs (Konno,H., et al. 1999)
  JOURNAL   Unpublished (1999)
COMMENT     Contact: Yoshihide Hayashizaki
            Laboratory for Genome Exploration Research Group, RIKEN Genomic
            Sciences Center(GSC), Yokohama Institute
            The Institute of Physical and Chemical Research (RIKEN)
            1-7-22 Suehiro-cho, Tsurumi-ku, Yokohama, Kanagawa 230-0045, Japan
            Tel: 81-45-503-9222
            Fax: 81-45-503-9216
            Email: genome-res@gsc.riken.go.jp
            URL:http://genome.gsc.riken.go.jp/
            Sasaki,N., Izawa,M., Watahiki,M., Ozawa,K., Tanaka,T., Yoneda,Y.,
            Matsuura,S., Carninci,P., Muramatsu,M., Okazaki,Y. and Hayashizaki
            ,Y.
            Transcriptional sequencing: A method for DNA sequencing using RNA
            polymerase. Proc. Natl. Acad. Sci. U.S.A. 95 (7), 3455-3460 (1998)
            Itoh,M., Kitsunai,T., Akiyama,J., Shibata,K., Izawa,M., Kawai,J.,
            Tomaru,Y., Carninci,P., Shibata,Y., Ozawa,Y., Muramatsu,M., Okazaki
            ,Y. and Hayashizaki,Y.
            Automated filtration-based high-throughput plasmid preparation
            system. Genome Res. 9 (5), 463-470 (1999)
            Carninci,P. and Hayashizaki,Y.
            High-efficiency full-length cDNA cloning. Methods Enzymol. 303,
            19-44 (1999)
            Please visit our web site (http://genome.rtc.riken.go.jp) for
            further details.
FEATURES             Location/Qualifiers
     source          1..195
                     /organism="Mus musculus"
                     /strain="C57BL/6J"
                     /db_xref="taxon:10090"
                     /clone="5530401C20"
                     /clone_lib="RIKEN full-length enriched, 10 days neonate
                     head"
                     /sex="mixed"
                     /tissue_type="head"
                     /dev_stage="10 days neonate"
                     /lab_host="DH10B"
                     /note="Site_1: SalI; Site_2: BamHI; cDNA library was
                     prepared and sequenced in Mouse Genome Encyclopedia
                     Project of Genome Exploration Research Group in Riken
                     Genomic Sciences Center and Genome Science Laboratory in
                     RIKEN. Division of Experimental Animal Research in Riken
                     contributed to prepare mouse tissues. 1st strand cDNA was
                     primed with a primer (5'
                     GAGAGAGAGAAGAGTCCAAGGAGCICITTTTTTTTTTTTTVN 3'). cDNA was
                     prepared by using trehalose thermo-activated reverse
                     transcriptase and subsequently enriched for full-length by
                     cap-trapper. cDNA went through one round of normalization
                     to Rot = 10.0 and subtraction to Rot = 100.0. Second
                     strand cDNA was prepared with the primer adapter of
                     sequence (5' GAGAGAGAGAATICGAGTCTAATTAAATTAACCCCCCCCCCCC
                     3'). cDNA was cloned into the XhoI and BamHI sites.
                     Vector: a modified pBluescript KS(+) after bulk excision
                     from lambda FLC I. Cloning sites, 5' end: SalI; 3' end:
                     BamHI."
BASE COUNT       40 a     43 c     39 g     73 t
ORIGIN


  Query Match             85.0%;  Score 17;  DB 9;  Length 195;
  Best Local Similarity   100.0%;  Pred. No. 6e+02;
  Matches   17;  Conservative    0;  Mismatches    0;  Indels    0;  Gaps    0;

Qy          2 ctgatattctggtggga 18
              |||||||||||||||||
Db         57 CTGATATTCTGGTGGGA 73


RESULT  10
BB067695
LOCUS       BB067695                 219 bp    mRNA    linear   EST 25-JUN-2002
DEFINITION  BB067695 RIKEN full-length enriched, 15 days embryo male testis Mus
            musculus cDNA clone B030466B06 3', mRNA sequence.
ACCESSION   BB067695
VERSION     BB067695.1  GI:8525109
KEYWORDS    EST.
SOURCE      house mouse.
  ORGANISM  Mus musculus
            Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
            Mammalia; Eutheria; Rodentia; Sciurognathi; Muridae; Murinae; Mus.
REFERENCE   1  (bases 1 to 219)
  AUTHORS   Konno,H., Aizawa,K., Akahira,S., Akiyama,J., Arakawa,T., Carninci
            ,P., Endo,T., Fukuda,S., Fukunishi,Y., Hara,A., Hayatsu,N.,
            Hirozane,T., Hori,F., Ishii,Y., Ishikawa,J., Ishikawa,T., Itoh,M.
            Izawa,M., Kadota,K., Kagawa,I., Kai,C., Kawai,J., Kikuchi,N.,
            Kojima,Y., Kondo,S., Koya,S., Kurihara,C., Kusakabe,M.
            , Matsuyama,T., Miki,R., Mizuno,Y., Nakamura,M., Oda,H., Okazaki,Y.
            , Owa,C., Saito,H., Sakai,C., Sato,K., Shibata,K., Shibata
            ,Y., Shigemoto,Y., Shinagawa,A., Shiraki,T., Sogabe,Y., Sugahara,Y
            , Suzuki,H., Suzuki,H., Tagawa,A., Takahashi,F., Tominaga,N., Toya
            ,T., Tsunoda,Y., Watahiki,A., Watanabe,S., Yamamura,T., Yamanaka,I.
            , Yano,R., Yasunishi,A., Yokota,T., Yoshida,K., Yoshiki,A., Yoshino
            ,M., Muramatsu,M. and Hayashizaki,Y.
  TITLE     RIKEN Mouse ESTs (Konno,H., et al.)
  JOURNAL   Unpublished (2000)
COMMENT     Contact: Yoshihide Hayashizaki
            Laboratory for Genome Exploration Research Group, RIKEN Genomic
            Sciences Center(GSC), Yokohama Institute
            The Institute of Physical and Chemical Research (RIKEN)
            1-7-22 Suehiro-cho, Tsurumi-ku, Yokohama, Kanagawa 230-0045, Japan
            Tel: 81-45-503-9222
            Fax: 81-45-503-9216
            Email: genome-res@gsc.riken.go.jp
            URL:http://genome.gsc.riken.go.jp/
            Carninci,P., Nishiyama,Y., Westover,A., Itoh,M., Nagaoka,S., Sasaki
            ,N., Okazaki,Y., Muramatsu,M. and Hayashizaki,Y.
```

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LO     34 CTGATATTCTGGTGGGA 50


RESULT 12
AQ819189/c
LOCUS       AQ819189                 452 bp    DNA     linear   GSS 26-AUG-1999
DEFINITION  HS_5300_A2_E07_SP6E RPCI-11 Human Male BAC Library Homo sapiens
            genomic clone Plate=876 Col=14 Row=I, DNA sequence.
ACCESSION   AQ819189
VERSION     AQ819189.1  GI:5781582
KEYWORDS    GSS.
SOURCE      human.
  ORGANISM  Homo sapiens
            Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
            Mammalia; Eutheria; Primates; Catarrhini; Hominidae; Homo.
REFERENCE   1  (bases 1 to 452)
  AUTHORS   Mahairas,G.G., Wallace,J.C., Smith,K., Swartzell,S., Holzman,I.,
            Keller,A., Shaker,R., Furlong,J., Young,J., Zhao,S., Adams,M.D. and
            Hood,L.
  TITLE     Sequence-tagged connectors: A sequence approach to mapping and
            scanning the human genome
  JOURNAL   Proc. Natl. Acad. Sci. U. S. A. 96 (17), 9739-9744 (1999)
  MEDLINE   99380589
  COMMENT   Contact: Mahairas GG, Wallace JC, Hood L
            High Throughput Sequencing Center
            University of Washington
            401 Queen Anne Avenue North, Seattle, WA 98109, USA
            Tel: (206) 616-3618
            Fax: (206) 616-3887
            Email: jwallace@u.washington.edu
            Clones are derived from the human BAC library RPCI-11. For BAC
            library availability, please contact Pieter de Jong
            (pieter@dejong.med.buffalo.edu). Clones may be purchased from
            BACPAC Resources (http://bacpac.med.buffalo.edu/ordering_bac.htm)
            or from Research Genetics (info@resgen.com). BAC end Web Server:
            http://www.htsc.washington.edu
            Plate: 876  row: I  column: 14
            Seq primer: SP6
            Class: BAC ends
            High quality sequence stop: 452.
FEATURES             Location/Qualifiers
     source          1..452
                     /organism="Homo sapiens"
                     /db_xref="taxon:9606"
                     /clone="Plate=876 Col=14 Row=I"
                     /clone_lib="RPCI-11 Human Male BAC Library"
                     /sex="male"
                     /note="Vector: pBACe3.6; Site_1: EcoRI; Site_2: EcoRI;
                     Male blood DNA was isolated from one randomly chosen donor
                     and partially digested with a combination of EcoRI and
                     EcoRI Methylase. Size selected DNA was cloned into the
                     pBACe3.6 vector at EcoRI sites"
BASE COUNT      147 a      66 c      89 g     148 t       2 others
ORIGIN

  Query Match           85.0%;  Score 17;  DB 12;  Length 452;
  Best Local Similarity  100.0%;  Pred. No. 6.9e+02;
  Matches   17;  Conservative    0;  Mismatches    0;  Indels    0;  Gaps    0;

Qy      4 gatattctggtgggaaa 20
          |||||||||||||||||
Db    366 GATATTCTGGTGGGAAA 350


RESULT 13
AQ437759/c
LOCUS       AQ437759                 519 bp    DNA     linear   GSS 31-MAR-1999
DEFINITION  HS_5056_B1_F03_T7A RPCI-11 Human Male BAC Library Homo sapiens
            genomic clone Plate=632 Col=5 Row=L, DNA sequence.
ACCESSION   AQ437759
VERSION     AQ437759.1  GI:4549098
KEYWORDS    GSS.
SOURCE      human.
  ORGANISM  Homo sapiens
            Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
            Mammalia; Eutheria; Primates; Catarrhini; Hominidae; Homo.
REFERENCE   1  (bases 1 to 519)
  AUTHORS   Mahairas,G.G., Wallace,J.C., Smith,K., Swartzell,S., Holzman,I.,
            Keller,A., Shaker,R., Furlong,J., Young,J., Zhao,S., Adams,M.D. and
            Hood,L.
  TITLE     Sequence-tagged connectors: A sequence approach to mapping and
            scanning the human genome
  JOURNAL   Proc. Natl. Acad. Sci. U. S. A. 96 (17), 9739-9744 (1999)
  MEDLINE   99380589
  COMMENT   Contact: Mahairas GG, Wallace JC, Hood L
            High Throughput Sequencing Center
            University of Washington
            401 Queen Anne Avenue North, Seattle, WA 98109, USA
            Tel: (206) 616-3618
            Fax: (206) 616-3887
            Email: jwallace@u.washington.edu
            Clones are derived from the human BAC library RPCI-11. For BAC
            library availability, please contact Pieter de Jong
            (pieter@dejong.med.buffalo.edu). Clones may be purchased from
            BACPAC Resources (http://bacpac.med.buffalo.edu/ordering_bac.htm)
            or from Research Genetics (info@resgen.com). BAC end Web Server:
            http://www.htsc.washington.edu
            Plate: 632  row: L  column: 5
            Seq primer: T7
            Class: BAC ends
            High quality sequence stop: 519.
FEATURES             Location/Qualifiers
     source          1..519
                     /organism="Homo sapiens"
                     /db_xref="taxon:9606"
                     /clone="Plate=632 Col=5 Row=L"
                     /clone_lib="RPCI-11 Human Male BAC Library"
                     /sex="male"
                     /note="Vector: pBACe3.6; Site_1: EcoRI; Site_2: EcoRI;
                     Male blood DNA was isolated from one randomly chosen donor
                     and partially digested with a combination of EcoRI and
                     EcoRI Methylase. Size selected DNA was cloned into the
                     pBACe3.6 vector at EcoRI sites"
BASE COUNT      141 a      85 c     103 g     165 t       5 others
ORIGIN

  Query Match           85.0%;  Score 17;  DB 12;  Length 519;
  Best Local Similarity  100.0%;  Pred. No. 7.1e+02;
  Matches   17;  Conservative    0;  Mismatches    0;  Indels    0;  Gaps    0;

Qy      4 gatattctggtgggaaa 20
          |||||||||||||||||
Db    382 GATATTCTGGTGGGAAA 366


RESULT 14
AQ420961
LOCUS       AQ420961                 132 bp    DNA     linear   GSS 23-MAR-1999
DEFINITION  RPCI-11-210I21.TV RPCI-11 Homo sapiens genomic clone RPCI-11-210I21
            , DNA sequence.
ACCESSION   AQ420961
VERSION     AQ420961.1  GI:4478705
KEYWORDS    GSS.
SOURCE      human.
  ORGANISM  Homo sapiens
            Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
            Mammalia; Eutheria; Primates; Catarrhini; Hominidae; Homo.
REFERENCE   1  (bases 1 to 132)
  AUTHORS   Zhao,S., Adams,M.D., Nierman,W., Malek,J., de Jong,P. and Venter
            ,J.C.
  TITLE     Use of BAC End Sequences from Library RPCI-11 for Sequence-Ready
            Map Building
  JOURNAL   Unpublished (1997)
  COMMENT   Contact: Shaying Zhao, William Nierman, Mark Adams
```

```
            Department of Eukaryotic Genomics
            The Institute for Genomic Research
            9712 Medical Center Dr., Rockville, MD 20850
            Tel: 301 838 0200
            Fax: 301 838 0208
            Email: hbm@tigr.org
            Clones are derived from the human BAC library RPCI-11. For BAC
            library availability, please contact Pieter de Jong
            (pieter@delong.med.buffalo.edu). Clones may be purchased from
            BACPAC Resources (http://bacpac.med.buffalo.edu/ordering) or from
            Research Genet cs (info@resgen.com). BAC end search page:
            http://www.tigr.org/tdb/humgen/bac_end_search/bac_end_search.html
            Seq primer: T7
            Class: BAC ends.
FEATURES             Location/Qualifiers
     source          1..132
                     /organism="Homo sapiens"
                     /db_xref="GDB:7580468"
                     /db_xref="taxon:9606"
                     /clone="RPCI-11-210121"
                     /clone_lib="RPCI-11"
                     /sex="Male"
                     /cell_type="Lymphocytes"
                     /note="Vector: pBACe3.6; Site_1: EcoRI; Site_2: EcoRI;
                     RPCI11 Human Male BAC Library"
BASE COUNT       43 a     19 c     44 g     26 t
ORIGIN


  Query Match             84.0%;  Score 16.8;  DB 12;  Length 132;
  Best Local Similarity   90.0%;  Pred. No. 7e-02;
  Matches   18;  Conservative    0;  Mismatches    2;  Indels    0;  Gaps    0;

Qy       1 gctgatattctggtgggaaa 20
           |||||| |||||||| ||||
Db       4 GCTGATCTTCTGGTGAGAAA 23


RESULT 15
BF758769/c
LOCUS       BF758769               154 bp    mRNA    linear   EST 12-JAN-2001
DEFINITION  MR0-CT0451-161100-019-f05 CT0451 Homo sapiens cDNA, mRNA sequence.
ACCESSION   BF758769
VERSION     BF758769.1  GI:12106669
KEYWORDS    EST.
SOURCE      human.
  ORGANISM  Homo sapiens
            Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
            Mammalia; Eutheria; Primates; Catarrhini; Hominidae; Homo.
REFERENCE   1  (bases 1 to 154)
  AUTHORS   Dias Neto,E., Garcia Correa,R., Verjovski-Almeida,S., Briones,M.R.,
            Nagai,M.A., da Silva,W. Jr., Zago,M.A., Bordin,S., Costa,F.F.,
            Goldman,G.H., Carvalho,A.F., Matsukuma,A., Baia,G.S., Simpson,D.H.,
            Brunstein,A., deOliveira,P.S., Bucher,P., Jonasmeel,C.V., O'Hare
            ,M.J., Soares,F., Brentani,R.R., Reis,L.F., de Souza,S.J. and
            Simpson,A.J.
  TITLE     Shotgun sequencing of the human transcriptome with ORF expressed
            sequence tags
  JOURNAL   Proc. Natl. Acad. Sci. U.S.A. 97 (7), 3491-3496 (2000)
  MEDLINE   20202663
COMMENT     Contact: Simpson A.J.G.
            Laboratory of Cancer Genetics
            Ludwig Institute for Cancer Research
            Rua Prof. Antonio Prudente 109, 4 andar, 01509-010, Sao Paulo-SP,
            Brazil
            Tel: -55-11-2704922
            Fax: -55-11-2707001
            Email: asimpson@ludwig.org.br
            This sequence was derived from the FAPESP/LICR Human Cancer Genome
            Project. This entry can be seen in the following URL
            (http://www.ludwig.org.br/scripts/gethtml2.pl?tl=MR0&t2=MR0-CT0451-
            161100-019-f05&t3=2000-11-16&t4=1)
            Seq primer: puc 18 forward
            High quality sequence stop: 153.
FEATURES             Location/Qualifiers
     source          1..154
                     /organism="Homo sapiens"
                     /db_xref="taxon:9606"
                     /clone_lib="CT0451"
                     /dev_stage="Adult"
                     /note="Organ: colon; Vector: puc18; Site_1: SmaI; Site_2:
                     SmaI; A mini-library was made by cloning products derived
                     from  ORESTES PCR (U.S. Letters Patent application No. 196
                     ,716 -  Ludwig Institute for Cancer Research) profiles
                     into  the pUC 18 vector. Reverse transcription of tissue
                     mRNA and cDNA amplification were performed under low
                     stringency conditions."
BASE COUNT       56 a     27 c     40 g     31 t
ORIGIN


  Query Match             84.0%;  Score 16.8;  DB 10;  Length 154;
  Best Local Similarity   90.0%;  Pred. No. 7.2e+02;
  Matches   18;  Conservative    0;  Mismatches    2;  Indels    0;  Gaps    0;

Qy       1 gctgatattctggtgggaaa 20
           |||||| |||||||| ||||
Db     118 GCTGATTTTCTTGTGGGAAA 99


Search completed: July 18, 2002, 04:22:41
Job time: 6815 sec
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          Thermostabilization and thermoactivation of thermolabile enzymes by
          trehalose and its application for the synthesis of full length
          cDNA. Proc. Natl. Acad. Sci. U.S.A. 95 (2), 520-524 (1998)
           Itoh,M., Kitsunai,T., Akiyama,J., Shibata,K., Izawa,M., Kawai,J.,
          Tomaru,Y., Carninci,P., Shibata,Y., Ozawa,Y., Muramatsu,M., Okazaki
          ,Y. and Hayashizaki,Y.
           Automated filtration-based high-throughput plasmid preparation
          system. Genome Res. 9 (5), 463-470 (1999)
           Carninci,P. and Hayashizaki,Y.
           High-efficiency full-length cDNA cloning. Methods Enzymol. 303,
          19-44 (1999)
           Please visit our web site (http://genome.rtc.riken.go.jp) for
          further details.
FEATURES             Location/Qualifiers
     source          1..219
                     /organism="Mus musculus"
                     /strain="C57BL/6J"
                     /db_xref="taxon:10090"
                     /clone="B030466H06"
                     /clone_lib="RIKEN full-length enriched, 15 days embryo
                     male testis"
                     /sex="male"
                     /tissue_type="testis"
                     /dev_stage="15 days embryo"
                     /lab_host="DH10B"
                     /note="Site_1: SalI; Site_2: BamHI; cDNA library was
                     prepared and sequenced in Mouse Genome Encyclopedia
                     Project of Genome Exploration Research Group in Riken
                     Genomic Sciences Center and Genome Science Laboratory in
                     RIKEN, Division of Experimental Animal Research in Riken
                     contributed to prepare mouse tissues. 1st strand cDNA was
                     primed with a primer [5'
                     GAGAGAGAGAAGGATCCAAGAGCTCTTTTTTTTTTTTTTVN 3'], cDNA was
                     prepared by using trehalose thermo-activated reverse
                     transcriptase and subsequently enriched for full-length by
                     cap-trapper. cDNA went through one round of subtraction to
                     Rot = 185.0 Second strand cDNA was prepared with the
                     primer adapter of sequence [5'
                     GAGAGAGAGATTCTCGAGTTAATTAAATTAATCCCCCCCCCCCC 3']. cDNA
                     was cloned into the XhoI and BamHI sites. Vector: a
                     modified pBluescript KS(+) after bulk excision from Lambda
                     FLC 1. Cloning sites, 5' end: SalI; 3' end: BamHI"
BASE COUNT        39 a      55 c      50 g      75 t
ORIGIN

  Query Match          85.0%;  Score 17;  DB 9;  Length 219;
  Best Local Similarity   100.0%;  Pred. No. 6.1e-02;
  Matches   17;  Conservative    0;  Mismatches    0;  Indels    0;  Gaps    0;

Qy       2 ctgatattctgtgtggg 18
           ||||||||||||||||||
Db      61 CTGATATTCTGGTGGGA 77


RESULT  11
BB047357
LOCUS       BB047357                 248 bp    mRNA    linear   EST 25-JUN-2000
DEFINITION  BB047357 RIKEN full-length enriched, adult male medulla oblongata
            Mus musculus cDNA clone 6330597K03 3', mRNA sequence.
ACCESSION   BB047357
VERSION     BB047357.1  GI:8454533
KEYWORDS    EST.
SOURCE      house mouse.
  ORGANISM  Mus musculus
            Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
            Mammalia; Eutheria; Rodentia; Sciurognathi; Muridae; Murinae; Mus
REFERENCE   1  (bases 1 to 248)
  AUTHORS   Konno,H., Aizawa,K., Akahira,S., Akiyama,J., Arakawa,T., Carninci
            ,P., Endo,T., Fukuda,S., Fukunishi,Y., Hara,A., Miyatani,N.,
            Hirotane,T., Hori,F., Ishii,Y., Ishikawa,J., Ishikawa,T., Itoh,M.,
            Izawa,M., Kadota,K., Kagawa,I., Kai,C., Kawai,J., Kikuchi,N.,
            Kiyosawa,H., Kojima,Y., Kondo,S., Koya,S., Kurihara,C., Kusakabe,M.
            , Matsuyama,T., Miki,R., Mizuno,Y., Nakamura,M., Oda,H., Okazaki,Y.
            , Ono,T., Owa,C., Saito,H., Sakai,C., Sato,K., Shibata,K., Shinta
            ,Y., Shigemoto,Y., Shinagawa,A., Shiraki,T., Sogabe,Y., Sugahara,Y.
            , Suzuki,H., Suzuki,H., Tagawa,A., Takahashi,F., Tominaga,N., Toya
            ,T., Tsunoda,Y., Watahiki,A., Watanabe,S., Yamamura,T., Yamanaki,T
            , Yano,R., Yasuoishi,A., Yokota,T., Yoshida,K., Yoshiki,A., Yoshimo
            ,M., Muramatsu,M. and Hayashizaki,Y.
  TITLE     RIKEN Mouse ESTs (Konno,H., et al.)
  JOURNAL   Unpublished (2000)
COMMENT     Contact: Yoshihide Hayashizaki
            Laboratory for Genome Exploration Research Group, RIKEN Genomic
            Sciences Center(GSC), Yokohama Institute
            The Institute of Physical and Chemical Research (RIKEN)
            1-7-22 Suehiro-cho, Tsurumi-ku, Yokohama, Kanagawa 230-0045, Japan
            Tel: 81-45-503-9222
            Fax: 81-45-503-9216
            Email: genome-res@gsc.riken.go.jp,
            URL:http://genome.gsc.riken.go.jp/
            Carninci,P., Nishiyama,Y., Westover,A., Itoh,M., Nagaoka,S., Sasaki
            ,N., Okazaki,Y., Muramatsu,M. and Hayashizaki,Y.
            Thermostabilization and thermoactivation of thermolabile enzymes by
            trehalose and its application for the synthesis of full length
            cDNA. Proc. Natl. Acad. Sci. U.S.A. 95 (2), 520-524 (1998)
             Itoh,M., Kitsunai,T., Akiyama,J., Shibata,K., Izawa,M., Kawai,J.,
            Tomaru,Y., Carninci,P., Shibata,Y., Ozawa,Y., Muramatsu,M., Okazaki
            ,Y. and Hayashizaki,Y.
             Automated filtration-based high-throughput plasmid preparation
            system. Genome Res. 9 (5), 463-470 (1999)
             Carninci,P. and Hayashizaki,Y.
             High-efficiency full-length cDNA cloning. Methods Enzymol. 303,
            19-44 (1999)
             Please visit our web site (http://genome.rtc.riken.go.jp) for
            further details.
FEATURES             Location/Qualifiers
     source          1..248
                     /organism="Mus musculus"
                     /strain="C57BL/6J"
                     /db_xref="taxon:10090"
                     /clone="6330597K03"
                     /clone_lib="RIKEN full-length enriched, adult male medulla
                     oblongata"
                     /sex="male"
                     /tissue_type="medulla oblongata"
                     /dev_stage="adult"
                     /lab_host="DH10B"
                     /note="Site_1: SalI; Site_2: BamHI; cDNA library was
                     prepared and sequenced in Mouse Genome Encyclopedia
                     Project of Genome Exploration Research Group in Riken
                     Genomic Sciences Center and Genome Science Laboratory in
                     RIKEN, Division of Experimental Animal Research in Riken
                     contributed to prepare mouse tissues. 1st strand cDNA was
                     primed with a primer [5'
                     GAGAGAGAGAAGGATCCAAGAGCTCTTTTTTTTTTTTTTVN 3'], cDNA was
                     prepared by using trehalose thermo-activated reverse
                     transcriptase and subsequently enriched for full-length by
                     cap-trapper. cDNA went through one round of normalization
                     to Rot = 10.0 and subtraction to Rot = 100.0. Second
                     strand cDNA was prepared with the primer adapter of
                     sequence [5' GAGAGAGAGATTCTCGAGTTAATTAAATTAATCCCCCCCCCCCC
                     3']. cDNA was cloned into the XhoI and BamHI sites.
                     Vector: a modified pBluescript KS(+) after bulk excision
                     from Lambda FLC 1. Cloning sites, 5' end: SalI; 3' end:
                     BamHI"
BASE COUNT        50 a      53 c      64 g      81 t
ORIGIN

  Query Match          85.0%;  Score 17;  DB 9;  Length 248;
  Best Local Similarity   100.0%;  Pred. No. 6.3e+02;
  Matches   17;  Conservative    0;  Mismatches    0;  Indels    0;  Gaps    0;

Qy       2 ctgatattctgtgtggg 18
           ||||||||||||||||||
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                Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
                Mammalia; Eutheria; Primates; Catarrhini; Hominidae; Pan.
REFERENCE   1  (sites)
  AUTHORS   Fujiyama,A., Hattori,M., Toyoda,A., Taylor,T.D., Yada,T.,
            Totoki,Y., Watanabe,H. and Sakaki,Y.
  TITLE     BAC end sequences of Library RPCI-43
  JOURNAL   Unpublished
REFERENCE   2  (bases 1 to 588)
  AUTHORS   Fujiyama,A., Hattori,M., Toyoda,A., Taylor,T.D., Yada,T.,
            Totoki,Y., Watanabe,H. and Sakaki,Y.
  TITLE     Direct Submission
  JOURNAL   Submitted (02-AUG-2001) Asao Fujiyama, The Institute of Physical
            and Chemical Research (RIKEN), Genomic Sciences Center (GSC),
            1-7-22 Suehiro-chou,Tsurumi-ku, Yokohama, Kanagawa 230-0045, Japan
            (E-mail:chimphes@gsc.riken.go.jp, URL:http://hgp.gsc.riken.go.jp/.
            Tel:81-45-503-9111, Fax:81-45-503-9170)
COMMENT     Clones are derived from the chimpanzee BAC library RPCI-43 This BAC
            end was generated during the R&D process and may have higher chance
            of clone tracking errors.
              PRIMERS
                  Sequencing: T7
              LIBRARY
                  Vector    : pBACe3.6
                  R.Site 1  : EcoRI
                  R.Site 2  : EcoRI.
FEATURES             Location/Qualifiers
     source          1..688
                     /organism="Pan troglodytes"
                     /db_xref="taxon:9598"
                     /clone="RP43-054D23.T7"
                     /sex="male"
                     /cell_type="lymphocytes"
                     /clone_lib="RPCI-43 Chimpanzee Male BAC Library"
BASE COUNT       242 a    115 c    127 g    201 t      2 others
ORIGIN

  Query Match           87.0%;  Score 17.4;  DB 12;  Length 688;
  Best Local Similarity   94.7%;  Pred. No. 4.8e-02;
  Matches   18;  Conservative    0;  Mismatches    1;  Indels    0;  Gaps    0;

Qy      2 ctgatattctggtgggaaa 20
          || ||||||||||||||||
Db    261 CTCATATTCTGGTGGGAAA 279


RESULT   7
AG181575
LOCUS       AG181575                 697 bp    DNA     linear   GSS 09-JAN-2002
DEFINITION  Pan troglodytes DNA, clone: RP43-054H23.T7, genomic survey
            sequence.
ACCESSION   AG181575
VERSION     AG181575.1  GI:16711255
KEYWORDS    GSS; GSS (genome survey sequence).
SOURCE      Pan troglodytes male lymphocytes DNA, clone_lib:RPCI-43 Chimpanzee
            Male BAC library clone:RP43-054H23.T7.
  ORGANISM  Pan troglodytes
            Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
            Mammalia; Eutheria; Primates; Catarrhini; Hominidae; Pan.
REFERENCE   1  (sites)
  AUTHORS   Fujiyama,A., Hattori,M., Toyoda,A., Taylor,T.D., Yada,T.,
            Totoki,Y., Watanabe,H. and Sakaki,Y.
  TITLE     BAC end sequences of Library RPCI-43
  JOURNAL   Unpublished
REFERENCE   2  (bases 1 to 697)
  AUTHORS   Fujiyama,A., Hattori,M., Toyoda,A., Taylor,T.D., Yada,T.,
            Totoki,Y., Watanabe,H. and Sakaki,Y.
  TITLE     Direct Submission
  JOURNAL   Submitted (02-AUG-2001) Asao Fujiyama, The Institute of Physical
            and Chemical Research (RIKEN), Genomic Sciences Center (GSC),
            1-7-22 Suehiro-chou,Tsurumi-ku, Yokohama, Kanagawa 230-0045, Japan
            (E-mail:chimphes@gsc.riken.go.jp, URL:http://hgp.gsc.riken.go.jp/.
            Tel:81-45-503-9111, Fax:81-45-503-9170)
COMMENT     Clones are derived from the chimpanzee BAC library RPCI-43 This BAC
            end was generated during the R&D process and may have higher chance
            of clone tracking errors.
              PRIMERS
                  Sequencing: T7
              LIBRARY
                  Vector    : pBACe3.6
                  R.Site 1  : EcoRI
                  R.Site 2  : EcoRI.
FEATURES             Location/Qualifiers
     source          1..697
                     /organism="Pan troglodytes"
                     /db_xref="taxon:9598"
                     /clone="RP43-054H23.T7"
                     /sex="male"
                     /cell_type="lymphocytes"
                     /clone_lib="RPCI-43 Chimpanzee Male BAC Library"
BASE COUNT       252 a    113 c    131 g    197 t      4 others
ORIGIN

  Query Match           87.0%;  Score 17.4;  DB 12;  Length 697;
  Best Local Similarity   94.7%;  Pred. No. 4.8e-02;
  Matches   18;  Conservative    0;  Mismatches    1;  Indels    0;  Gaps    0;

Qy      2 ctgatattctggtgggaaa 20
          || ||||||||||||||||
Db    260 CTCATATTCTGGTGGGAAA 278


RESULT   8
AV011849
LOCUS       AV011849                 176 bp    mRNA    linear   EST 25-AUG-1999
DEFINITION  AV011849 Mus musculus 18-day embryo C57BL/6J Mus musculus cDNA
            clone 1110033K13, mRNA sequence.
ACCESSION   AV011849
VERSION     AV011849.1  GI:4788836
KEYWORDS    EST.
SOURCE      house mouse.
  ORGANISM  Mus musculus
            Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
            Mammalia; Eutheria; Rodentia; Sciurognathi; Muridae; Murinae; Mus.
REFERENCE   1  (bases 1 to 176)
  AUTHORS   Carninci,P., Shibata,K., Ozawa,Y., Konno,H., Itoh,M., Aizawa,K.,
            Akahira,S., Akiyama,J., Fukuda,S., Fukunishi,Y., Funayama,T., Hara
            ,A., Hayatsu,N., Hori,F., Ishikawa,T., Itoh,M., Izawa,M., Kawai,J.,
            Kikuchi,N., Kojima,Y., Matsuyama,T., Nitasuma,H., Oda,H., Owa,C.,
            Sato,K., Shibata,Y., Shigemoto,Y., Shiraki,T., Sogabe,Y., Sugahara
            ,Y., Suzuki,H., Suzuki,H., Tateno,M., Tomaru,Y., Tominaga,N.,
            Watanabe,S., Yagame,M., Yamamura,T., Yokota,T., Yoshino,M.,
            Kuramatsu,M., Okazaki,Y. and Hayashizaki,Y.
  TITLE     RIKEN Mouse ESTs
  JOURNAL   Unpublished (1999)
COMMENT     Contact: Chie Owa
            Genome Science Laboratory
            RIKEN
            3-1-1 Koyadai, Tsukuba, Ibaraki 305-0074, Japan
            Tel: 81-298-36-9145
            Fax: 81-298-36-9098
            Email: genome-res@rtc.riken.go.jp
            Thermostabilization and thermoactivation of thermolabile enzymes by
            trehalose and its application for the synthesis of full length cDNA
            (Proc. Natl. Acad. Sci. U.S.A. 95(2):520-524 (1998))
            Transcriptional sequencing: A method for DNA sequencing using RNA
            polymerase (Proc. Natl. Acad. Sci. U.S.A. 95(7):3455-3460 (1998))
            Please visit our web site (http://genome.rtc.riken.go.jp) for
            further details.
FEATURES             Location/Qualifiers
     source          1..176
                     /organism="Mus musculus"
                     /strain="C57BL/6J"
                     /db_xref="taxon:10090"
                     /clone="1110033K13"
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                     /db_xref="taxon:9606"
                     /clone="Plate=643 Col=5 Row=E"
                     /clone_lib="RPCI-11 Human Male BAC Library"
                     /sex="male"
                     /note="Vector: pBAC3.6; Site_1: EcoRI; Site_2: EcoRI;
                     Male blood DNA was isolated from one randomly chosen donor
                     and partially digested with a combination of EcoRI and
                     EcoRI Methylase. Size selected DNA was cloned into the
                     pBAC3.6 vector at EcoRI sites"
BASE COUNT       119 a    136 c    105 g    138 t      5 others
ORIGIN


  Query Match             90.0%;  Score 18;  DB 12;  Length 523;
  Best Local Similarity   100.0%;  Pred. No. 2.4e-02;
  Matches   18;  Conservative    0;  Mismatches    0;  Indels    0;  Gaps    0;

Qy          2 ctgtattctggtgggaa 19
              ||||||||||||||||||
Db        518 CTGATATTCTGGTGGGAA 501


RESULT   2
BM188182/c
LOCUS       BM188182                 551 bp    mRNA    linear   EST 11-DEC-2001
DEFINITION  sa85f07.y1 Gm-c1074 Glycine max cDNA clone SOYBEAN CLONE ID:
            Gm-c1074-3350 5' similar to TP:Q9ZT66 Q9ZT66 ENDO-1,3-1
            ,4-BETA-D-GLUCANASE. ; mRNA sequence.
ACCESSION   BM188182
VERSION     BM188182.1  GI:17519140
KEYWORDS    EST.
SOURCE      soybean.
  ORGANISM  Glycine max
            Eukaryota; Viridiplantae; Streptophyta; Embryophyta; Tracheophyta;
            Spermatophyta; Magnoliophyta; eudicotyledons; core eudicots;
            Rosidae; eurosids I; Fabales; Fabaceae; Papilionideae; Phaseoleae;
            Glycine.
REFERENCE   1  (bases 1 to 551)
  AUTHORS   Shoemaker,R., Keim,P., Vodkin,L., Erpelding,J., Cotyell V., Khanna
            ,A., Bolla,R., Marra,M., Hillier,L., Kucaba,T., Martin J., Beck,C.,
            Wylie,T., Underwood,K., Steptoe,M., Theising,B., Allen,M., Bowers
            ,Y., Person,B., Swaller,T., Gibbons,M., Pape,D., Harvey,N., Schurk
            ,R., Bitter,E., Kohn,S., Shin,T., Jackson,Y., Cardenas,M., McCann
            ,K., Waterston,R. and Wilson,R.
  TITLE     Public Soybean EST Project
  JOURNAL   Unpublished (1999)
COMMENT     Contact: Shoemaker R/Public Soybean EST Project
            Public Soybean EST Project
            Washington University School of Medicine
            4444 Forest Park Parkway, Box 8501, St. Louis, MO 63108, USA
            Tel: 314 286 1800
            Fax: 314 286 1810
            Email: est@watson.wustl.edu
            This clone is available through: ResGen, Invitrogen Corp, 2130
            South Memorial Parkway Huntsville, AL 35801 For further information
            call: (800)-533-4363 or contact: ccutreesgen.com Web site:
            www.resgen.com
            Seq primer: -40PF from Gibco
            High quality sequence stop: 430.
FEATURES             Location/Qualifiers
     source          1..551
                     /organism="Glycine max"
                     /db_xref="taxon:3847"
                     /clone="SOYBEAN CLONE ID: Gm-c1074-3350"
                     /clone_lib="Gm-c1074"
                     /tissue_type="seedlings induced for HR (hypersensitive
                     response)"
                     /dev_stage="9-11 day old"
                     /lab_host="DH10B"
                     /note="Vector: pBluescript II SK-; Site_1: EcoRI; Site_2:
                     XhoI: The cDNA library was constructed from mRNA isolated
                     from 9-11 day old seedlings that were induced for HR
                     (hypersensitive response) by vacuum infiltrating plant
                     tissue with Pseudomonas syringae pv. glycinea carrying the
                     avrB gene (Genetics 141:1597-1604). Plant tissue (expanded
                     unifoliate leaves) was collected at 2, 4, 8, 12, 24, 36,
                     and 53 hrs after inoculation and their mRNA pooled equally
                     for cDNA construction. The library was prepared using the
                     Stratagene pBluescript II SK(+) library construction kit.
                     Complementary DNA was synthesized from mRNA using a primer
                     consisting of a poly(dT) sequence with an XhoI restriction
                     site. EcoRI adaptors were ligated to the blunt-ended cDNA
                     fragments followed by XhoI digestion. The cDNA insert is
                     protected from XhoI digestion via methylation during first
                     strand synthesis. The cDNA fragments were directionally
                     cloned into the EcoRI-XhoI restriction site of the
                     pBluescript vector. The ligated cDNA fragments were
                     transformed into E.coli ElectroMax DH10B host cells. Plant
                     care, inoculations, and library construction were
                     performed by Steve Clough (Lila Vodkin lab, University of
                     Illinois)."
BASE COUNT       148 a    110 c    126 g    167 t
ORIGIN


  Query Match             90.0%;  Score 18;  DB 10;  Length 551;
  Best Local Similarity   100.0%;  Pred. No. 2.4e-02;
  Matches   18;  Conservative    0;  Mismatches    0;  Indels    0;  Gaps    0;

Qy          3 tgtattctggtgggaaga 20
              ||||||||||||||||||
Db        536 TGATATTCTGGTGGGAAA 519


RESULT   3
BE913740
LOCUS       BE913740                 729 bp    mRNA    linear   EST 29-SEP-2000
DEFINITION  601669205F1 NCI_CGAP_Mam1 Mus musculus cDNA clone IMAGE:3969121 5',
            mRNA sequence.
ACCESSION   BE913740
VERSION     BE913740.1  GI:10411662
KEYWORDS    EST.
SOURCE      house mouse
  ORGANISM  Mus musculus
            Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
            Mammalia; Eutheria; Rodentia; Sciurognathi; Muridae; Murinae; Mus.
REFERENCE   1  (bases 1 to 729)
  AUTHORS   NIH-MGC http://mgc.nci.nih.gov/.
  TITLE     National Institutes of Health, Mammalian Gene Collection (MGC)
  JOURNAL   Unpublished (1999)
COMMENT     Contact: Robert Strausberg, Ph.D.
            Email: cgapbs-r@mail.nih.gov
            Tissue Procurement: Gilbert Smith, Ph.D.
            cDNA Library Preparation: Life Technologies, Inc.
            cDNA Library Arrayed by: The I.M.A.G.E. Consortium (LLNL)
            DNA Sequencing by: Incyte Genomics, Inc.
            Clone distribution: MGC clone distribution information can be
            found through the I.M.A.G.E. Consortium/LLNL at:
            http://image.llnl.gov
            Plate: LLAM9146  row: j  column: 02
            High quality sequence stop: 724.
FEATURES             Location/Qualifiers
     source          1..729
                     /organism="Mus musculus"
                     /strain="FVB/N"
                     /db_xref="taxon:10090"
                     /clone="IMAGE:3969121"
                     /clone_lib="NCI_CGAP_Mam1"
                     /tissue_type="tumor, biopsy sample"
                     /dev_stage="10 months, virgin"
                     /lab_host="DH10B"
                     /note="Organ: mammary; Vector: pCMV-SPORT6; Site_1: SalI;
                     Site_2: NotI; Cloned unidirectionally.  Primer: Oligo dT.
                     Library constructed by Life Technologies. Investigator
                     providing samples: Gilbert Smith, NIH"
BASE COUNT       152 a    182 c    236 g    159 t
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;       ZIP:  20850
;     COMPUTER READABLE FORM:
;       MEDIUM TYPE:  Floppy disk
;       COMPUTER:  IBM PC compatible
;       OPERATING SYSTEM:  PC-DOS/MS-DOS
;       SOFTWARE:  PatentIn Release #1.0, Version #1.30
;     CURRENT APPLICATION DATA:
;       APPLICATION NUMBER:  US/09/002,072B
;       FILING DATE:  09-FEB-1998
;       CLASSIFICATION:  435
;     PRIOR APPLICATION DATA:
;       APPLICATION NUMBER:  US 08/461,731
;       FILING DATE:  05-JUN-1995
;     PRIOR APPLICATION DATA:
;       APPLICATION NUMBER:  WO PCT/US94/10465
;       FILING DATE:  16-SEP-1994
;     ATTORNEY/AGENT INFORMATION:
;       NAME:  Brookes, A. Anders
;       REGISTRATION NUMBER:  36,373
;       REFERENCE/DOCKET NUMBER:  PF134D1
;     TELECOMMUNICATION INFORMATION:
;       TELEPHONE:  301-309-8504
;       TELEFAX:  301-309-8439
;   INFORMATION FOR SEQ ID NO:  3:
;     SEQUENCE CHARACTERISTICS:
;       LENGTH:  1313 base pairs
;       TYPE:  nucleic acid
;       STRANDEDNESS:  single
;       TOPOLOGY:  linear
;     MOLECULE TYPE:  DNA (genomic)
US-09-002-072B-3


  Query Match             74.0%;  Score 14.8;  DB 2;  Length 1313;
  Best Local Similarity   88.9%;  Pred. No. 80;
  Matches   16;  Conservative    0;  Mismatches    2;  Indels    0;  Gaps    0;

Qy       3 tgatattctgatgggaaa 20
           | ||||||||| ||||||
Db    1202 TIATATICIGGGGGAAA 1185


Search completed: July 18, 2002, 04:56:38
Job time: 8267 sec
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```
;    APPLICANT:  Philippsen, Peter
;    APPLICANT:  Pohlmann, Reiner
;    APPLICANT:  Stelner, Sabine
;    APPLICANT:  Mohr, Christine
;    APPLICANT:  Wendland, Jurgen
;    APPLICANT:  Knechtle, Philipp
;    APPLICANT:  Rebischung, Corinne
;    TITLE OF INVENTION:  GENOMIC DNA SEQUENCES OF ASHBYA GOSSYPII
;    TITLE OF INVENTION:  AND USES THEREOF
;    NUMBER OF SEQUENCES:  5152
;    CORRESPONDENCE ADDRESS:
;      ADDRESSEE:  No. 6239264artis Corporation
;      STREET:  3054 Cornwallis Road
;      CITY:  Research Triangle Park
;      STATE:  No. 6239264th Carolina
;      COUNTRY:  USA
;      ZIP:  27709
;    COMPUTER READABLE FORM:
;      MEDIUM TYPE:  Floppy disk
;      COMPUTER:  IBM PC compatible
;      OPERATING SYSTEM:  PC-DOS/MS-DOS
;      SOFTWARE:  PatentIn Release #1.0, Version #1.30
;    CURRENT APPLICATION DATA:
;      APPLICATION NUMBER:  US/08/998,416
;      FILING DATE:  24-DEC-1997
;      CLASSIFICATION:  435
;    PRIOR APPLICATION DATA:
;      APPLICATION NUMBER:  CH 0016/97
;      FILING DATE:  31-DEC-1996
;    ATTORNEY/AGENT INFORMATION:
;      NAME:  Meigs, J. Timothy
;      REGISTRATION NUMBER:  38,241
;      REFERENCE/DOCKET NUMBER:  PF/5-30306/A/CGC1976
;    TELECOMMUNICATION INFORMATION:
;      TELEPHONE:  919-541-8587
;      TELEFAX:  919-541-8689
; INFORMATION FOR SEQ ID NO:  900:
;    SEQUENCE CHARACTERISTICS:
;      LENGTH:  711 base pairs
;      TYPE:  nucleic acid
;      STRANDEDNESS:  single
;      TOPOLOGY:  linear
;    MOLECULE TYPE:  DNA (genomic)
;    ORIGINAL SOURCE:
;      ORGANISM:  PAC1561UP
US-08-998-416-900

  Query Match           76.0%;  Score 15.2;  DB 4;  Length 711;
  Best Local Similarity   85.0%;  Pred. No. 45;
  Matches  17;  Conservative    0;  Mismatches    3;  Indels    0;  Gaps    0;

Qy      1 gctgctattctggtggcaaaa 20
          |||||  |||||||||| |||
Db     96 GCTGACCTTCTGGTGCCAAA 115


RESULT 11
US-08-772-440-33
; Sequence 33, Application US/08772440
; Patent No. 6046159
;  GENERAL INFORMATION:
;    APPLICANT:  Arilzumi, Kiyoshi
;    APPLICANT:  Takeshima, Akira
;    TITLE OF INVENTION:  UNIQUE DENDRITIC CELL-ASSOCIATED C-TYPE
;    TITLE OF INVENTION:  LECTINS, DECTIN-1 AND DECTIN-2: COMPOSITIONS AND USES
;    TITLE OF INVENTION:  THEREOF
;    NUMBER OF SEQUENCES:  42
;    CORRESPONDENCE ADDRESS:
;      ADDRESSEE:  Arnold, White & Durkee
;      STREET:  P.O. Box 4433
;      CITY:  Houston
;      STATE:  Texas
;      COUNTRY:  USA
;      ZIP:  77210
;    COMPUTER READABLE FORM:
;      MEDIUM TYPE:  Floppy disk
;      COMPUTER:  IBM PC compatible
;      OPERATING SYSTEM:  PC-DOS/MS-DOS
;      SOFTWARE:  PatentIn Release #1.0, Version #1.30
;    CURRENT APPLICATION DATA:
;      APPLICATION NUMBER:  US/08/772,440
;      FILING DATE:  CONCURRENTLY HEREWITH
;      CLASSIFICATION:  435
;    ATTORNEY/AGENT INFORMATION:
;      NAME:  Parker, David L.
;      REGISTRATION NUMBER:  32,165
;      REFERENCE/DOCKET NUMBER:  UTXD:493
;    TELECOMMUNICATION INFORMATION:
;      TELEPHONE:  512/418-3000
;      TELEFAX:  512/474-7577
; INFORMATION FOR SEQ ID NO:  33:
;    SEQUENCE CHARACTERISTICS:
;      LENGTH:  10409 base pairs
;      TYPE:  nucleic acid
;      STRANDEDNESS:  single
;      TOPOLOGY:  linear
;    FEATURE:
;      NAME/KEY:  modified_base
;      LOCATION:  6510
;      OTHER INFORMATION:  /mod_base= OTHER
;      OTHER INFORMATION:  /note= "D = A or G or T"
;    FEATURE:
;      NAME/KEY:  modified_base
;      LOCATION:  3106..6470
;      OTHER INFORMATION:  /mod_base= OTHER
;      OTHER INFORMATION:  /note= "K = G or T"
;    FEATURE:
;      NAME/KEY:  modified_base
;      LOCATION:  3564..7896
;      OTHER INFORMATION:  /mod_base= OTHER
;      OTHER INFORMATION:  /note= "M = A or C"
;    FEATURE:
;      NAME/KEY:  modified_base
;      LOCATION:  3497..3607
;      OTHER INFORMATION:  /mod_base= OTHER
;      OTHER INFORMATION:  /note= "N = A or C or G or T"
;    FEATURE:
;      NAME/KEY:  modified_base
;      LOCATION:  3479..6422
;      OTHER INFORMATION:  /mod_base= OTHER
;      OTHER INFORMATION:  /note= "R = A or G"
;    FEATURE:
;      NAME/KEY:  modified_base
;      LOCATION:  3405..6871
;      OTHER INFORMATION:  /mod_base= OTHER
;      OTHER INFORMATION:  /note= "S = C or G"
;    FEATURE:
;      NAME/KEY:  modified_base
;      LOCATION:  3457..9998
;      OTHER INFORMATION:  /mod_base= OTHER
;      OTHER INFORMATION:  /note= "W = A or T"
;    FEATURE:
;      NAME/KEY:  modified_base
;      LOCATION:  3595..9999
;      OTHER INFORMATION:  /mod_base= OTHER
;      OTHER INFORMATION:  /note= "Y = C or T"
US-08-772-440-33

  Query Match           76.0%;  Score 15.2;  DB 3;  Length 10409;
  Best Local Similarity   85.0%;  Pred. No. 68;
  Matches  17;  Conservative    0;  Mismatches    3;  Indels    0;  Gaps    0;

Qy      1 gctgctattctggtggcaaaa 20
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; MOLECULE TYPE: DNA (genomic)
US-09-032-742-3

Query Match 82.0%; Score 16.4; DB 4; Length 2246;
Best Local Similarity 94.4%; Pred. No. 13;
Matches 17; Conservative 0; Mismatches 1; Indels 0; Gaps 0;

Qy 1 gctgatattctgtggga 18
||||||| |||||||||
Db 982 GCTGATATGCTGGTGGGA 999

RESULT 5
US-09-032-742-21
; Sequence 21, Application US/09032742
; Patent No. 6255089
; GENERAL INFORMATION:
; APPLICANT: Teitler, Milt
; APPLICANT: Herrick-Davis, Katharine
; APPLICANT: Egan, Christina C.
; TITLE OF INVENTION: Constitutively Activated Serotonin
; TITLE OF INVENTION: Receptors
; NUMBER OF SEQUENCES: 25
; CORRESPONDENCE ADDRESS:
; ADDRESSEE: Laurence Weinberger
; STREET: 882 S. Matlack Street, Suite 103
; STREET: P.O. Box 1663
; CITY: West Chester
; STATE: PA
; COUNTRY: USA
; ZIP: 19380-0053
; COMPUTER READABLE FORM:
; MEDIUM TYPE: Floppy disk
; COMPUTER: IBM PC compatible
; OPERATING SYSTEM: PC-DOS/MS-DOS
; SOFTWARE: PatentIn Release #1.0, Version #1.30
; CURRENT APPLICATION DATA:
; APPLICATION NUMBER: US/09/032,742
; FILING DATE: 27-FEB-1998
; CLASSIFICATION: 536
; ATTORNEY/AGENT INFORMATION:
; NAME: Weinberger, Laurence
; REGISTRATION NUMBER: 27,965
; REFERENCE/DOCKET NUMBER: 3086-4
; TELECOMMUNICATION INFORMATION:
; TELEPHONE: (610) 431-1703
; TELEFAX: (610) 431-4181
; INFORMATION FOR SEQ ID NO: 21:
; SEQUENCE CHARACTERISTICS:
; LENGTH: 2246 base pairs
; TYPE: nucleic acid
; STRANDEDNESS: single
; TOPOLOGY: linear
; MOLECULE TYPE: DNA (genomic)
US-09-032-742-21

Query Match 82.0%; Score 16.4; DB 4; Length 2246;
Best Local Similarity 94.4%; Pred. No. 13;
Matches 17; Conservative 0; Mismatches 1; Indels 0; Gaps 0;

Qy 1 gctgatattctgtggga 18
||||||| |||||||||
Db 982 GCTGATATGCTGGTGGGA 999

RESULT 6
US-09-032-742-22
; Sequence 22, Application US/09032742
; Patent No. 6255089
; GENERAL INFORMATION:
; APPLICANT: Teitler, Milt
; APPLICANT: Herrick-Davis, Katharine
; APPLICANT: Egan, Christina C.
; TITLE OF INVENTION: Constitutively Activated Serotonin
; TITLE OF INVENTION: Receptors
; NUMBER OF SEQUENCES: 25
; CORRESPONDENCE ADDRESS:
; ADDRESSEE: Laurence Weinberger
; STREET: 882 S. Matlack Street, Suite 103
; STREET: P.O. Box 1663
; CITY: West Chester
; STATE: PA
; COUNTRY: USA
; ZIP: 19380-0053
; COMPUTER READABLE FORM:
; MEDIUM TYPE: Floppy disk
; COMPUTER: IBM PC compatible
; OPERATING SYSTEM: PC-DOS/MS-DOS
; SOFTWARE: PatentIn Release #1.0, Version #1.30
; CURRENT APPLICATION DATA:
; APPLICATION NUMBER: US/09/032,742
; FILING DATE: 27-FEB-1998
; CLASSIFICATION: 536
; ATTORNEY/AGENT INFORMATION:
; NAME: Weinberger, Laurence
; REGISTRATION NUMBER: 27,965
; REFERENCE/DOCKET NUMBER: 3086-4
; TELECOMMUNICATION INFORMATION:
; TELEPHONE: (610) 431-1703
; TELEFAX: (610) 431-4181
; INFORMATION FOR SEQ ID NO: 22:
; SEQUENCE CHARACTERISTICS:
; LENGTH: 2246 base pairs
; TYPE: nucleic acid
; STRANDEDNESS: single
; TOPOLOGY: linear
; MOLECULE TYPE: DNA (genomic)
US-09-032-742-22

Query Match 82.0%; Score 16.4; DB 4; Length 2246;
Best Local Similarity 94.4%; Pred. No. 13;
Matches 17; Conservative 0; Mismatches 1; Indels 0; Gaps 0;

Qy 1 gctgatattctgtggga 18
||||||| |||||||||
Db 982 GCTGATATGCTGGTGGGA 999

RESULT 7
US-09-032-742-24
; Sequence 24, Application US/09032742
; Patent No. 6255089
; GENERAL INFORMATION:
; APPLICANT: Teitler, Milt
; APPLICANT: Herrick-Davis, Katharine
; APPLICANT: Egan, Christina C.
; TITLE OF INVENTION: Constitutively Activated Serotonin
; TITLE OF INVENTION: Receptors
; NUMBER OF SEQUENCES: 25
; CORRESPONDENCE ADDRESS:
; ADDRESSEE: Laurence Weinberger
; STREET: 882 S. Matlack Street, Suite 103
; STREET: P.O. Box 1663
; CITY: West Chester
; STATE: PA
; COUNTRY: USA
; ZIP: 19380-0053
; COMPUTER READABLE FORM:
; MEDIUM TYPE: Floppy disk
; COMPUTER: IBM PC compatible
; OPERATING SYSTEM: PC-DOS/MS-DOS
; SOFTWARE: PatentIn Release #1.0, Version #1.30
; CURRENT APPLICATION DATA:

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      STREET:  4 Embarcadero Center, Suite 3400
      CITY:  San Francisco
      STATE:  California
      COUNTRY:  USA
      ZIP:  94111
    COMPUTER READABLE FORM:
      MEDIUM TYPE:  Floppy disk
      COMPUTER:  IBM PC compatible
      OPERATING SYSTEM:  PC-DOS/MS-DOS
      SOFTWARE:  PatentIn Release #1.0, Version #1.25
    CURRENT APPLICATION DATA:
      APPLICATION NUMBER:  US/08/313,553
      FILING DATE:
      CLASSIFICATION:  435
    PRIOR APPLICATION DATA:
      APPLICATION NUMBER:  US/08/038,662
      FILING DATE:
    ATTORNEY/AGENT INFORMATION:
      NAME:  Dreger, Walter H.
      REGISTRATION NUMBER:  24,190
      REFERENCE/DOCKET NUMBER:  A-57669/WHD
    TELECOMMUNICATION INFORMATION:
      TELEPHONE:  (415) 781-1989
      TELEFAX:  (415) 398-3249
  INFORMATION FOR SEQ ID NO:  10:
    SEQUENCE CHARACTERISTICS:
      LENGTH:  1848 base pairs
      TYPE:  nucleic acid
      STRANDEDNESS:  single
      TOPOLOGY:  linear
    MOLECULE TYPE:  cDNA
    FEATURE:
      NAME/KEY:  misc_feature
      LOCATION:  376..414
      OTHER INFORMATION:  /note= "Bacteriorhodopsin
      OTHER INFORMATION:  pre-sequence."
    FEATURE:
      NAME/KEY:  terminator
      LOCATION:  1756..1758
      OTHER INFORMATION:  /note= "Bacteriorhodopsin stop
      OTHER INFORMATION:  codon."
    FEATURE:
      NAME/KEY:  misc_feature
      LOCATION:  517..591
      OTHER INFORMATION:  /note= "Helix I of rat serotoni
      OTHER INFORMATION:  receptor protein (Type 1C)."
    FEATURE:
      NAME/KEY:  misc_feature
      LOCATION:  625..690
      OTHER INFORMATION:  /note= "Helix II of rat serotonin
      OTHER INFORMATION:  receptor protein (Type 1C)."
    FEATURE:
      NAME/KEY:  misc_feature
      LOCATION:  736..807
      OTHER INFORMATION:  /note= "Helix III of rat serotonin
      OTHER INFORMATION:  receptor protein (Type 1C)."
    FEATURE:
      NAME/KEY:  misc_feature
      LOCATION:  868..939
      OTHER INFORMATION:  /note= "Helix IV of rat serotonin
      OTHER INFORMATION:  receptor protein (Type 1C)."
    FEATURE:
      NAME/KEY:  misc_feature
      LOCATION:  997..1059
      OTHER INFORMATION:  /note= "Helix V of rat serotonin
      OTHER INFORMATION:  receptor protein (Type 1C)."
    FEATURE:
      NAME/KEY:  misc_feature
      LOCATION:  1297..1362
      OTHER INFORMATION:  /note= "Helix VI of rat serotonin
      OTHER INFORMATION:  receptor protein (Type 1C)."
    FEATURE:
      NAME/KEY:  misc_feature
      LOCATION:  1411..1476
      OTHER INFORMATION:  /note= "Helix VII of rat serotonin
      OTHER INFORMATION:  receptor protein (Type 1C)."
    FEATURE:
      NAME/KEY:  mutation
      LOCATION:  replace(213, "")
      OTHER INFORMATION:  /note= "G to A mutation removes
      OTHER INFORMATION:  AlwNI restriction site."
    FEATURE:
      NAME/KEY:  misc_feature
      LOCATION:  1732..1734
      OTHER INFORMATION:  /note= "Codon encoding the
      OTHER INFORMATION:  C-terminal amino acid of the rat serotonin
      OTHER INFORMATION:  receptor protein (Type 1C)."
    FEATURE:
      NAME/KEY:  misc_signal
      LOCATION:  374
      OTHER INFORMATION:  /note= "RNA start site."
    FEATURE:
      NAME/KEY:  CDS
      LOCATION:  376..1734
    FEATURE:
      NAME/KEY:  terminator
      LOCATION:  1735..1737
      OTHER INFORMATION:  /note= "Serotonin stop codon."
    FEATURE:
      NAME/KEY:  repeat_region
      LOCATION:  436..462
      OTHER INFORMATION:  /note= "Sequence encoding
Patent No. 5641650
      OTHER INFORMATION:  polyaspartic acid."
    FEATURE:
      NAME/KEY:  mutation
      LOCATION:  replace(1755, "")
      OTHER INFORMATION:  /note= "C to T mutation removes
      OTHER INFORMATION:  AlwNI restriction site."
US-08-313-553-10

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  Best Local Similarity   94.4%;  Pred. No. 13;
  Matches   17;  Conservative    0;  Mismatches    1;  Indels    0;  Gaps    0;

Qy       1 gctgatattctgatggga 18
           ||||||||| ||||||||||
Db     649 GCTGATATGCTGGTGGGA 666


RESULT   3
US-08-767-993-10
; Sequence 10, Application US/08767993
; Patent No. 6010885
;  GENERAL INFORMATION:
;    APPLICANT:  TURNER, George J.
;    APPLICANT:  BETLACH, Mary C.
;    TITLE OF INVENTION:  EXPRESSION OF HETEROLOGOUS POLYPEPTIDES
;    TITLE OF INVENTION:  IN HALOBACTERIA
;    NUMBER OF SEQUENCES:  15
;    CORRESPONDENCE ADDRESS:
;      ADDRESSEE:  Walter H. Dreger
;      STREET:  4 Embarcadero Center, Suite 3400
;      CITY:  San Francisco
;      STATE:  California
;      COUNTRY:  USA
;      ZIP:  94111
;    COMPUTER READABLE FORM:
;      MEDIUM TYPE:  Floppy disk
;      COMPUTER:  IBM PC compatible
;      OPERATING SYSTEM:  PC-DOS/MS-DOS
;      SOFTWARE:  PatentIn Release #1.0, Version #1.25
;    CURRENT APPLICATION DATA:
;      APPLICATION NUMBER:  US/08/767,993
;      FILING DATE:
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DP   P-PSDB; ABG24634.
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PT   New isolated polynucleotide and encoded polypeptides, useful in
PT   diagnostics, forensics, gene mapping, identification of mutations
PT   responsible for genetic disorders or other traits and to assess
PT   biodiversity -
XX
PS   Claim 1; SEQ ID No 24625; 103pp; English.
XX
CC   The invention relates to isolated polynucleotide (I) and
CC   polypeptide (II) sequences. (I) is useful as hybridisation probes,
CC   polymerase chain reaction (PCR) primers, oligomers, and for chromosome
CC   and gene mapping, and in recombinant production of (II). The
CC   polynucleotides are also used in diagnostics as expressed sequence tags
CC   for identifying expressed genes. (I) is useful in gene therapy techniques
CC   to restore normal activity of (II) or to treat disease states involving
CC   (II). (II) is useful for generating antibodies against it, detecting or
CC   quantitating a polypeptide in tissue, as molecular weight markers and as
CC   a food supplement. (II) and its binding partners are useful in medical
CC   imaging of sites expressing (II). (I) and (II) are useful for treating
CC   disorders involving aberrant protein expression or biological activity.
CC   The polypeptide and polynucleotide sequences have applications in
CC   diagnostics, forensics, gene mapping, identification of mutations
CC   responsible for genetic disorders or other traits to assess biodiversity
CC   and to produce other types of data and products dependent on DNA and
CC   amino acid sequences. AAS64197-AAS94564 represent novel human
CC   diagnostic coding sequences of the invention.
CC   Note: The sequence data for this patent did not appear in the printed
CC   specification, but was obtained in electronic format directly from WIPO
CC   at ftp.wipo.int/pub/published_pct_sequences.
XX
SQ   Sequence 2469 BP; 575 A; 668 C; 704 G; 521 T; 1 other;

  Query Match             84.0%;  Score 16.8;  DB 23;  Length 2469;
  Best Local Similarity   90.0%;  Pred. No. 69;
  Matches   18;  Conservative    0;  Mismatches    2;  Indels    0;  Gaps    0;

Qy       1 gctgatattctggtgggaaa 20
           || ||||||||||||| ||
Db     121 gcggatattctggtgggtaa 140


RESULT  15
AAS88825
ID   AAS88825 standard; cDNA; 2829 BP.
XX
AC   AAS88825;
XX
DT   13-FEB-2002  (first entry)
XX
DE   DNA encoding novel human diagnostic protein #24629.
XX
KW   Human; chromosome mapping; gene mapping; gene therapy; forensic;
KW   food supplement; medical imaging; diagnostic; genetic disorder; ss
XX
OS   Homo sapiens.
XX
PN   WO200175067-A2.
XX
PD   11-OCT-2001.
XX
PF   30-MAR-2001; 2001WO-US08631.
XX
PR   31-MAR-2000; 2000US-0540217.
PR   23-AUG-2000; 2000US-0649167.
XX
PA   (HYSE-) HYSEQ INC.
XX
PI   Drmanac RT, Liu C, Tang YT;
XX
DR   WPI; 2001-639352/73.
DR   P-PSDB; ABG24633.
XX
PT   New isolated polynucleotide and encoded polypeptides, useful in
PT   diagnostics, forensics, gene mapping, identification of mutations
PT   responsible for genetic disorders or other traits and to assess
PT   biodiversity -
XX
PS   Claim 1; SEQ ID No 24629; 103pp; English.
XX
CC   The invention relates to isolated polynucleotide (I) and
CC   polypeptide (II) sequences. (I) is useful as hybridisation probes,
CC   polymerase chain reaction (PCR) primers, oligomers, and for chromosome
CC   and gene mapping, and in recombinant production of (II). The
CC   polynucleotides are also used in diagnostics as expressed sequence tags
CC   for identifying expressed genes. (I) is useful in gene therapy techniques
CC   to restore normal activity of (II) or to treat disease states involving
CC   (II). (II) is useful for generating antibodies against it, detecting or
CC   quantitating a polypeptide in tissue, as molecular weight markers and as
CC   a food supplement. (II) and its binding partners are useful in medical
CC   imaging of sites expressing (II). (I) and (II) are useful for treating
CC   disorders involving aberrant protein expression or biological activity.
CC   The polypeptide and polynucleotide sequences have applications in
CC   diagnostics, forensics, gene mapping, identification of mutations
CC   responsible for genetic disorders or other traits to assess biodiversity
CC   and to produce other types of data and products dependent on DNA and
CC   amino acid sequences. AAS64197-AAS94564 represent novel human
CC   diagnostic coding sequences of the invention.
CC   Note: The sequence data for this patent did not appear in the printed
CC   specification, but was obtained in electronic format directly from WIPO
CC   at ftp.wipo.int/pub/published_pct_sequences.
XX
SQ   Sequence 2829 BP; 644 A; 741 C; 823 G; 619 T; 2 other;

  Query Match             84.0%;  Score 16.8;  DB 23;  Length 2829;
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Db     949 gcggatattctggtgggtaa 968


Search completed: July 18, 2002, 05:03:04
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XX
PI   Drmanac PT,  Liu C,  Tang YT;
XX
DR   WPI; 2001-639362/73.
DR   P-PSDB; ABG24633.
XX
PT   New isolated polynucleotide and encoded polypeptides, useful in
PT   diagnostics, forensics, gene mapping, identification of mutations
PT   responsible for genetic disorders or other traits and to assess
PT   biodiversity -
XX
PS   Claim 1; SEQ ID No 24624; 103pp; English.
XX
CC   The invention relates to isolated polynucleotide (I) and
CC   polypeptide (II) sequences. (I) is useful as hybridisation probes,
CC   polymerase chain reaction (PCR) primers, oligomers, and for chromosome
CC   and gene mapping, and in recombinant production of (II). The
CC   polynucleotides are also used in diagnostics as expressed sequence tags
CC   for identifying expressed genes. (I) is useful in gene therapy techniques
CC   to restore normal activity of (II) or to treat disease states involving
CC   (II). (II) is useful for generating antibodies against it, detecting or
CC   quantitating a polypeptide in tissue, as molecular weight markers and as
CC   a food supplement. (II) and its binding partners are useful in medical
CC   imaging of sites expressing (II). (I) and (II) are useful for treating
CC   disorders involving aberrant protein expression or biological activity.
CC   The polypeptide and polynucleotide sequences have applications in
CC   diagnostics, forensics, gene mapping, identification of mutations
CC   responsible for genetic disorders or other traits to assess biodiversity
CC   and to produce other types of data and products dependent on DNA and
CC   amino acid sequences. AAS64197-AAS94564 represent novel human
CC   diagnostic coding sequences of the invention.
CC   Note: The sequence data for this patent did not appear in the printed
CC   specification, but was obtained in electronic format directly from WIPO
CC   at ftp.wipo.int/pub/published_pct_sequences.
XX
SQ   Sequence 486 BP; 92 A; 117 C; 161 G; 116 T; 0 other;

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Qy       1 gctgatattctgqtgggaaa 20
           || |||||||||||||| ||
Db     184 gcggatattctggtgggtaa 203


RESULT  11
AAS88827
ID   AAS88827 standard; cDNA; 2347 BP.
XX
AC   AAS88827;
XX
DT   13-FEB-2002  (first entry)
XX
DE   DNA encoding novel human diagnostic protein #24631.
XX
KW   Human; chromosome mapping; gene mapping; gene therapy; forensic;
KW   food supplement; medical imaging; diagnostic; genetic disorder; ss.
XX
OS   Homo sapiens.
XX
PN   WO200175067-A2.
XX
PD   11-OCT-2001.
XX
PF   30-MAR-2001; 2001WO-US08631.
XX
PR   31-MAR-2000; 2000US-0540217.
PR   23-AUG-2000; 2000US-0649167.
XX
PA   (HYSE-) HYSEQ INC.
XX
PI   Drmanac RT,  Liu C,  Tang YT;
XX
DR   WPI; 2001-639362/73.
DR   P-PSDB; ABG24640.
XX
PT   New isolated polynucleotide and encoded polypeptides, useful in
PT   diagnostics, forensics, gene mapping, identification of mutations
PT   responsible for genetic disorders or other traits and to assess
PT   biodiversity -
XX
PS   Claim 1; SEQ ID No 24631; 103pp; English.
XX
CC   The invention relates to isolated polynucleotide (I) and
CC   polypeptide (II) sequences. (I) is useful as hybridisation probes,
CC   polymerase chain reaction (PCR) primers, oligomers, and for chromosome
CC   and gene mapping, and in recombinant production of (II). The
CC   polynucleotides are also used in diagnostics as expressed sequence tags
CC   for identifying expressed genes. (I) is useful in gene therapy techniques
CC   to restore normal activity of (II) or to treat disease states involving
CC   (II). (II) is useful for generating antibodies against it, detecting or
CC   quantitating a polypeptide in tissue, as molecular weight markers and as
CC   a food supplement. (II) and its binding partners are useful in medical
CC   imaging of sites expressing (II). (I) and (II) are useful for treating
CC   disorders involving aberrant protein expression or biological activity.
CC   The polypeptide and polynucleotide sequences have applications in
CC   diagnostics, forensics, gene mapping, identification of mutations
CC   responsible for genetic disorders or other traits to assess biodiversity
CC   and to produce other types of data and products dependent on DNA and
CC   amino acid sequences. AAS64197-AAS94564 represent novel human
CC   diagnostic coding sequences of the invention.
CC   Note: The sequence data for this patent did not appear in the printed
CC   specification, but was obtained in electronic format directly from WIPO
CC   at ftp.wipo.int/pub/published_pct_sequences.
XX
SQ   Sequence 2347 BP; 551 A; 600 C; 697 G; 499 T; 0 other;

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Db    1172 gcggatattctggtgggtaa 1191


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ID   AAS88575/c standard; cDNA; 2370 BP.
XX
AC   AAS88575;
XX
DT   13-FEB-2002  (first entry)
XX
DE   DNA encoding novel human diagnostic protein #24379.
XX
KW   Human; chromosome mapping; gene mapping; gene therapy; forensic;
KW   food supplement; medical imaging; diagnostic; genetic disorder; ss.
XX
OS   Homo sapiens.
XX
PN   WO200175067-A2.
XX
PD   11-OCT-2001.
XX
PF   30-MAR-2001; 2001WO-US08631.
XX
PR   31-MAR-2000; 2000US-0540217.
PR   23-AUG-2000; 2000US-0649167.
XX
PA   (HYSE-) HYSEQ INC.
XX
PI   Drmanac RT,  Liu C,  Tang YT;
```

```
RESULT   6
AAF27670
ID   AAF27670 standard; DNA; 20 BP.
XX
AC   AAF27670;
XX
DT   02-APR-2001  (first entry)
XX
DE   Primer #3.
XX
KW   IL-1; interleukin; inflammation; infection; ss.
XX
OS   Unidentified.
XX
PN   WO200100880-A2.
XX
PD   04-JAN-2001.
XX
PF   30-JUN-2000; 2000WO-US18318.
XX
PR   30-JUN-1999;   99US-0345217.
XX
PA   (INTE-) INTERLEUKIN GENETICS INC.
XX
PI   Duff GW,  Cox A,  Camp NJ,  Di Giovine FS;
XX
DR   WPI; 2001-102903/11.
XX
PT   Determining whether a subject has or is predisposed to disease
PT   associated with IL-1 polymorphism involves determining presence of
PT   marker or allele comprising IL-1 inflammatory haplotype -
XX
PS   Disclosure: Page 23; 84pp; English.
XX
CC   The present invention relates to a new method for determining whether
CC   a subject has or is predisposed to developing a disease or condition
CC   that is associated with an IL (interleukin)-1 inflammatory haplotype,
CC   comprises detecting at least one allele of the haplotype, where the
CC   presence of the allele indicates that the subject is predisposed to
CC   the development or has the disease or condition.
CC   The method is useful for determining whether a subject has or is
CC   predisposed to inflammatory disease, a degenerative disease, an
CC   immunological disorder, an infectious disease, trauma induced disease,
CC   or cancer. The above conditions associated with an IL-1 inflammatory
CC   haplotype can be treated or prevented by administering a therapeutic
CC   that compensates for a causative mutation that is in linkage
CC   disequilibrium with at least one IL-1 polymorphism.
XX
SQ   Sequence 20 BP; 5 A; 2 C; 7 G; 6 T; 0 other;

  Query Match             100.0%;  Score 20;  DB 22;  Length 20;
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  Matches   20;  Conservative    0;  Mismatches    0;  Indels    0;  Gaps    0;

Qy       1 gctgatattctggtgggaaa 20
           ||||||||||||||||||||
Db       1 gctgatattctggtgggaaa 20


RESULT   7
AAC91438
ID   AAC91438 standard; DNA; 20 BP.
XX
AC   AAC91438;
XX
DT   20-MAR-2001  (first entry)
XX
DE   Human IL-1A polymorphic locus AFM087xa1 5' PCR primer.
XX
KW   Human; IL-1A; interleukin-1alpha; IL-1B; interleukin-1beta; IL-1RN;
KW   interleukin-1 receptor antagonist; vasotropic; antiinflammatory;
KW   hypotensive; anticoagulant; antilipaemic; arterial restenosis;
KW   restenosis associated allele; RAA; occlusive cardiovascular disorder;
KW   restenosis detection; PCR primer; ss.
XX
OS   Homo sapiens.
XX
PN   WO200071753-A2.
XX
PD   30-NOV-2000.
XX
PF   24-MAY-2000; 2000WO-US14299.
XX
PR   24-MAY-1999;   99US-0317674.
PR   01-NOV-1999;   99US-0431352.
XX
PA   (INTE-) INTERLEUKIN GENETICS INC.
XX
PI   Kornman KS,  Duff GW,  Crossman DC,  Francis SE,  Stephenson K;
XX
DR   WPI; 2001-025173/03.
XX
PT   Diagnosing or determining susceptibility to developing restenosis
PT   involves detecting restenosis associated allele in a nucleic acid
PT   sample -
XX
PS   Disclosure: Page 41; 129pp; English.
XX
CC   The present sequence is given in a specification relating to a method for
CC   determining whether a subject has or is predisposed to developing an
CC   arterial restenosis. The method comprises detecting a restenosis
CC   associated allele (RAA) in a nucleic acid sample from the subject, where
CC   detection of the RAA indicates that the subject has or is predisposed to
CC   the development of a restenosis. The restenosis associated allelic
CC   pattern permits the diagnosis of occlusive cardiovascular disorder. The
CC   diagnosis allows the most suitable treatment methods for restenosis to be
CC   used e.g. selecting therapies for initial vascular stenosis most likely
CC   to avoid subsequent stenoses. The detection methods identify restenosis
CC   therapeutics, agonists and antagonists, (proteins, peptides,
CC   peptidomimetics, small molecules or nucleic acids, e.g. anti-sense,
CC   ribozyme and triplex nucleic acids) which are used to treat restenosis
XX
SQ   Sequence 20 BP; 5 A; 2 C; 7 G; 6 T; 0 other;

  Query Match             100.0%;  Score 20;  DB 22;  Length 20;
  Best Local Similarity   100.0%;  Pred. No. 0.89;
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Db       1 gctgatattctggtgggaaa 20


RESULT   8
AAC89183
ID   AAC89183 standard; DNA; 20 BP.
XX
AC   AAC89183;
XX
DT   08-MAR-2001  (first entry)
XX
DE   Human microsatellite marker AFM087xa1 PCR primer #1.
XX
KW   Human; PCR primer; cardiant; vasotropic; polymorphic; chromosome 2q13;
KW   interleukin-1 gene cluster; IL-1; cardiovascular disorder;
KW   fragile plaque disorder; occlusive disorder; in-stent restenosis; ss.
XX
OS   Homo sapiens.
XX
PN   WO200072015-A2.
XX
PD   30-NOV-2000.
XX
PF   26-MAY-2000; 2000WO-US14775.
```

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XX
PS   Claim 9; Page 8; 21pp; English.
XX
CC   PCR primers AAZ28417-Z28418 are used to amplify the microsatellite
CC   markers associated with the allele situated on chromosome 2, containing
CC   the D2S121 locus. The D2S121 microsatellite markers are contained in the
CC   region of chromosome 2 containing the IL2 (interleukin 2) cluster of
CC   genes. The invention relates to a method for diagnosing asthma or a
CC   predisposition to asthma. The products of PCR primers AAZ28409-Z28410
CC   are used to detect any alleles that may be connected with asthma. The
CC   D2S308*2 allele (PCR primers AAZ28409-Z28410 are used to amplify the
CC   associated microsatellite markers) is used in the claimed methods to
CC   identify children at risk of developing asthma by examination
CC   immediately after birth, potentially allowing the disease to be
CC   prevented. The methods may also allow a prognosis of the severity of a
CC   condition and responses to particular treatments.
XX
SQ   Sequence 20 BP; 5 A; 2 C; 7 G; 6 T; 0 other;

  Query Match             100.0%;  Score 20;  DB 20;  Length 20;
  Best Local Similarity   100.0%;  Pred. No. 0.89;
  Matches   20;  Conservative    0;  Mismatches   0;  Indels    0;  Gaps    0;

Qy       1 gctgatattctgtgtaggaaa 20
           ||||||||||||||||||||
Db       1 gctgatattctgtgtaggaaa 20


RESULT   2
AAC63773
ID   AAC63773 standard; DNA; 20 BP.
XX
AC   AAC63773;
XX
DT   08-FEB-2001  (first entry)
XX
DE   Human IL-1 gene 5' PCR primer.
XX
KW   Human; IL-1; interleukin-1A; cytostatic; antiinflammatory;
KW   immunosuppressive; dermatological; antimicrobial; antiarthritic;
KW   IL-1 receptor antagonist; tumour necrosis factor alpha antagonist;
KW   interstitial lung disease; interstitial pneumonia; pulmonary fibrosis;
KW   rheumatoid arthritis; systemic lupus erythmatosis; Sjogren's syndrome;
KW   systemic sclerosis; dermatomyocitis; PCR primer; chromosome 2; ss.
XX
OS   Homo sapiens.
XX
PN   WO200060177-A2.
XX
PD   12-OCT-2000.
XX
PF   31-MAR-2000; 2000WO-US08492.
XX
PR   02-APR-1999;  99US-0286108.
XX
PA   (INTE-) INTERLEUKIN GENETICS INC.
XX
PI   Duff GW,  Di Giovine FS,  Whyte M;
XX
DR   WPI; 2000-656234/63.
XX
PT   Method for predicting the risk of interstitial lung disease, comprising
PT   detecting an interleukin-1 receptor antagonist allele and tumor
PT   necrosis alpha allele or an allele in linkage disequilibrium with
PT   either of these alleles -
XX
PS   Claim 6; Page 40; 102pp; English.
XX
CC   The present sequence is provided in a specification relating to a method
CC   for determining whether a subject has or is predisposed to develop an
CC   interstitisl lung disease. The method involves detecting an interleukin-1
CC   receptor antagonist (IL-1RN) (-2018) allele 2, a tumour necrosis alpha
CC   (TNF-A308) allele 2, or an allele in linkage disequilibrium with
CC   either of these two alleles. The method may be used to determine whether
CC   a subject has or is predisposed to develop an interstitial pneumonia or a
CC   pulmonary fibrosis and other disorders such as rheumatoid arthritis,
CC   systemic lupus erythmatosis, Sjogren's syndsome, systemic sclerosis,
CC   dermatomyocitis. The method is also used for identifying molecules
CC   which can be used as therapeutics for treating interstitial lung disease.
XX
SQ   Sequence 20 BP; 5 A; 2 C; 7 G; 6 T; 0 other;

  Query Match             100.0%;  Score 20;  DB 21;  Length 20;
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           ||||||||||||||||||||
Db       1 gctgatattctgtgtaggaaa 20


RESULT   1
AAA61927
ID   AAA61927 standard; DNA; 20 BP.
XX
AC   AAA61927;
XX
DT   20-NOV-2000  (first entry)
XX
DE   Human IL 1 gene cluster AFM087xa1 marker PCR primer, SEQ ID NO:13.
XX
KW   Human; IL-1 gene cluster; interleukin-1; chromosome 2q13-14;
KW   proinflammatory; cytokine; systemic inflammatory response; sepsis;
KW   dysregulation; pattern 2 allele; genotyping; centromere distal;
KW   polymorphic microsatellite anchor marker; AFM087xa1; PCR primer; ss.
XX
OS   Homo sapiens.
XX
PN   WO200037679-A2.
XX
PD   29-JUN-2000.
XX
PF   01-NOV-1999;  99WO-US25633.
XX
PR   30-OCT-1998;  98US-0183850.
XX
PA   (INTE-) INTERLEUKIN GENETICS INC.
XX
PI   Di Giovine FS,  Duff GW;
XX
DR   WPI; 2000-442691/38.
XX
PT   Determining susceptibility to developing sepsis for preventing or
PT   treating sepsis, involves detecting an allele of interleukin-1 genetic
PT   pattern that leads to dysregulated inflammatory response -
XX
PS   Disclosure; Page 36; 66pp; English.
XX
CC   The invention relates to methods of determining an individual's
CC   susceptibility to developing and/or rapidly progressing into sepsis via
CC   genotyping of a member of the IL-1 (interleukin-1) gene cluster. The
CC   method comprises obtaining an IL-1 gene cluster nucleic acid from an
CC   individual and determining whether that individual's DNA contains at
CC   least one allele of an IL-1 genetic pattern that leads to a dysregulated
CC   inflammatory response. The presence of a pattern 2 allele or a marker in
CC   linkage disequilibrium with a pattern 2 allele in the sample, indicates
CC   that the patient has an increased susceptibility to developing sepsis.
CC   The IL-1 gene cluster comprises a region of 430 kb which is located on
CC   the long arm of chromosome 2 (2q13-14). It contains the IL-1A gene which
CC   encodes IL-1-alpha, the IL-1B gene which encodes IL-1-beta, and the
CC   IL-1RN gene which encodes the IL-1 receptor antagonist. IL-1 is a
CC   proinflammatory cytokine released by macrophages which can induce a
CC   systemic response to local injury or infection. It thus plays a key role
CC   in the development of sepsis, which can lead to intravascular
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COMMENT     On May 20, 2001 this sequence version replaced gi:7321474.
            All repeats were identified using RepeatMasker
            Smit, A.F.A. & Green, P. (1996-1997)
            http://ftp.genome.washington.edu/RM/RepeatMasker.html
            -------------- Genome Center
                Center: Whitehead Institute/ MIT Center for Genome Research
                Center code: WIBR
                Web site: http://www-seq.wi.mit.edu
                Contact: sequence_submissions@genome.wi.mit.edu
            -------------- Project Information
                Center project name: L1026
                Center clone name: 463_D_17
            -------------- Summary Statistics
                Sequencing vector: M13; M77815; 58% of reads
                Sequencing vector: Plasmid: L08752; 2% of reads
                Sequencing vector: Plasmid; n/a; 40% of reads
                Chemistry: Dye-primer-amershan; 32% of reads
                Chemistry: Dye-terminator Big Dye; 68% of reads
                Assembly program: Phrap; version 0.960731
                Consensus quality: 194152 bases at least Q40
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                Consensus quality: 195410 bases at least Q20
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                Insert size: 195548.
            * NOTE: This is a 'working draft' sequence. It currently
            * consists of 4 contigs. The true order of the pieces
            * is not known and their order in this sequence record is
            * arbitrary. Gaps between the contigs are represented as
            * runs of N, but the exact sizes of the gaps are unknown.
            * This record will be updated with the finished sequence
            * as soon as it is available and the accession number will
            * be preserved.
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ORIGIN


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Search completed: July 18, 2002, 04:55:17
Job time: 9466 sec
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ORIGIN


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  Best Local Similarity   95.0%;  Pred. No. 39;
  Matches   19;  Conservative    0;  Mismatches    1;  Indels    0;  Gaps    0;

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VERSION     AC009108.9  GI:13786306
KEYWORDS    HTG; HTGS_PHASE2; HTGS_DRAFT; HTGS_ACTIVEFIN.
SOURCE      Human.
  ORGANISM  Homo sapiens
            Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
            Mammalia; Eutheria; Primates; Catarrhini; Hominidae; Homo.
REFERENCE   1  (bases 1 to 170481)
  AUTHORS   DOE Joint Genome Institute.
  TITLE     Sequencing of Human Chromosome 16
  JOURNAL   Unpublished
REFERENCE   2  (bases 1 to 170481)
  AUTHORS   DOE Joint Genome Institute.
  TITLE     Direct Submission
  JOURNAL   Submitted (03-AUG-1999) Production Sequencing Facility, DOE Joint
            Genome Institute, 2800 Mitchell Drive, Walnut Creek, CA 94598, USA
COMMENT     On Apr 25, 2001 this sequence version replaced gi:9964740.
            --------Genome Center
            Center: Joint Genome Institute
            Center Code: JGI
            Web site: http://www.jgi.doe.gov
            -------
            Project Information
            Center Project Name: 584502
            Center clone name: RPC1-11_463G9
            -------
            Summary Statistics
            Consensus quality: 167345 bases at least Q40
            Consensus quality: 169423 bases at least Q30
            Consensus quality: 169900 bases at least Q20
            Estimated insert size: 181440; agarose-fp estimation
            Estimated insert size: 170331; sum-of-contigs estimation
            Quality coverage:  10.33 in Q20 bases; agarose-fp estimation
            Quality coverage:  11.0 in Q20 bases; sum-of-contigs estimation.
            * NOTE: This is a 'working draft' sequence.  It currently
            * consists of 4 contigs. Gaps between the contigs
            * are represented as runs of N. The order of the pieces
            * is believed to be correct as given, however the sizes
            * of the gaps between them are based on estimates that have
            * provided by the submittor.
            * This sequence will be replaced
            * by the finished sequence as soon as it is available and
            * the accession number will be preserved.
            *      1    24870: contig of 24870 bp in length
            *  24871    24970: gap of unknown length
            *  24971    37912: contig of 12942 bp in length
```

```
            *    47874    48569: contig of 696 bp in length
            *    48570 48669: gap of      100 bp
            *    48670    49329: contig of 660 bp in length
            *    49330 49429: gap of      100 bp
            *    49430    50112: contig of 683 bp in length
            *    50113 50212: gap of      100 bp
            *    50213    50836: contig of 624 bp in length
            *    50837 50936: gap of      100 bp
            *    50937    51616: contig of 680 bp in length
            *    51617 51716: gap of      100 bp
            *    51717    52397: contig of 681 bp in length
            *    52398 52497: gap of      100 bp
            *    52498    53185: contig of 688 bp in length
            *    53186 53285: gap of      100 bp

  Query Match             92.0%;  Score 18.4;  DB 2;  Length 66227;
  Best Local Similarity   95.0%;  Pred. No. 41;
  Matches  19;  Conservative    0;  Mismatches    1;  Indels    0;  Gaps    0;

Qy      1 gctaatattctggtggaaaa 20
          ||| ||||||||||||||||
Db  40098 GCTTATATTCTGGTGGGAAA 40079



RESULT  11
AC079836
LOCUS       AC079836               153903 bp    DNA     linear   HTG 24-NOV-2000
DEFINITION  Homo sapiens chromosome 2 clone RP11-99K18, WORKING DRAFT SEQUENCE,
            25 unordered pieces.
ACCESSION   AC079836
VERSION     AC079836.3  GI:11323401
KEYWORDS    HTG; HTGS_PHASE1; HTGS_DRAFT.
SOURCE      human.
  ORGANISM  Homo sapiens
            Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
            Mammalia; Eutheria; Primates; Catarrhini; Hominidae; Homo.
REFERENCE   1  (bases 1 to 153903)
  AUTHORS   Waterston,R.H.
  TITLE     The sequence of Homo sapiens clone
  JOURNAL   Unpublished
REFERENCE   2  (bases 1 to 153903)
  AUTHORS   Waterston,R.H.
  TITLE     Direct Submission
  JOURNAL   Submitted (12-SEP-2000) Genome Sequencing Center, Washington
            University School of Medicine, 4444 Forest Park Parkway, St. Louis,
            MO 63108, USA
COMMENT     On Nov 24, 2000 this sequence version replaced gi:10179389.

            ---------------- Genome Center ----------------
            Center: Washington University Genome Sequencing Center
            Center code: WUGSC
            Web site:http://genome.wustl.edu/gsc/index.shtml
            -------------- Project Information --------------
            Center project name: H_NH0099K18
            -------------- Summary Statistics --------------
            Sequencing vector: M13; 94%
            Sequencing vector: plasmid: 0%
            Chemistry: Dye-primer ET: 94% of reads
            Chemistry: Dye-terminator Big Dye: 0% of reads
            Assembly program: Phrap; version 0.990319
            Consensus quality: 140203 bases at least Q40
            Consensus quality: 145183 bases at least Q30
            Consensus quality: 147598 bases at least Q20
            Insert size: 170000; agarose-fp
            Insert size: 151503; sum-of-contigs
            Quality coverage: 3.69 in Q20 bases; agarose-fp
            Quality coverage: 4.24 in Q20 bases; sum-of-contigs
            ------------------------------------------------

            * NOTE: This is a 'working draft' sequence. It currently
            * consists of 25 contigs. The true order of the pieces
            * is not known and their order in this sequence record is
            * arbitrary. Gaps between the contigs are represented as
            * runs of N, but the exact sizes of the gaps are unknown.
            * This record will be updated with the finished sequence
            * as soon as it is available and the accession number will
            * be preserved.
            *       1     1830: contig of 1830 bp in length
            *    1831     1930: gap of unknown length
            *    1931     3310: contig of 1380 bp in length
            *    3311     3410: gap of unknown length
            *    3411     5464: contig of 2054 bp in length
            *    5465     5564: gap of unknown length
            *    5565     7171: contig of 1607 bp in length
            *    7172     7271: gap of unknown length
            *    7272     9874: contig of 2603 bp in length
            *    9875     9974: gap of unknown length
            *    9975    11983: contig of 2009 bp in length
            *   11984    12083: gap of unknown length
            *   12084    14275: contig of 2192 bp in length
            *   14276    14375: gap of unknown length
            *   14376    17628: contig of 3253 bp in length
            *   17629    17728: gap of unknown length
            *   17729    21724: contig of 3996 bp in length
            *   21725    21824: gap of unknown length
            *   21825    25332: contig of 3508 bp in length
            *   25333    25432: gap of unknown length
            *   25433    27718: contig of 2286 bp in length
            *   27719    27818: gap of unknown length
            *   27819    30797: contig of 2979 bp in length
            *   30798    30897: gap of unknown length
            *   30898    35000: contig of 4103 bp in length
            *   35001    35100: gap of unknown length
            *   35101    40321: contig of 5221 bp in length
            *   40322    40421: gap of unknown length
            *   40422    45944: contig of 5523 bp in length
            *   45945    46044: gap of unknown length
            *   46045    51804: contig of 5760 bp in length
            *   51805    51904: gap of unknown length
            *   51905    58214: contig of 6310 bp in length
            *   58215    58314: gap of unknown length
            *   58315    65112: contig of 6798 bp in length
            *   65113    65212: gap of unknown length
            *   65213    74226: contig of 9014 bp in length
            *   74227    74326: gap of unknown length
            *   74327    82657: contig of 8331 bp in length
            *   82658    82757: gap of unknown length
            *   82758    91941: contig of 9184 bp in length
            *   91942    92041: gap of unknown length
            *   92042   102415: contig of 10374 bp in length
            *  102416   102515: gap of unknown length
            *  102516   115951: contig of 13436 bp in length
            *  115952   116051: gap of unknown length
            *  116052   131428: contig of 15377 bp in length
            *  131429   131528: gap of unknown length
            *  131529   153903: contig of 22375 bp in length.
FEATURES             Location/Qualifiers
     source          1..153903
                     /organism="Homo sapiens"
                     /db_xref="taxon:9606"
                     /chromosome="2"
                     /clone="RP11-99K18"
     misc_feature    1..1830
                     /note="assembly_name:Contig12"
     misc_feature    1931..3310
                     /note="assembly_name:Contig13
                     clone_end:T7
                     vector_side:right"
     misc_feature    3411..5464
                     /note="assembly_name:Contig14"
     misc_feature    5565..7171
                     /note="assembly_name:Contig15"
     misc_feature    7272..9874
                     /note="assembly_name:Contig16"
     misc_feature    9975..11983
                     /note="assembly_name:Contig17"
     misc_feature    12084..14275
```

```
  Query Match             100.0%;  Score 20;  DB 2;  Length 185276;
  Best Local Similarity   100.0%;  Pred. No. 5.4;
  Matches   20;  Conservative    0;  Mismatches    0;  Indels    0;  Gaps    0;

Qy       1 gctgatatttctggtgggaaa 20
           ||||||||||||||||||||
Db   22816 GCTGATATTCTGGTGGGAAA 22797


RESULT   9
AL627261/c
LOCUS       AL627261               147777 bp    DNA     linear   HTG 19-DEC-2001
DEFINITION  Danio rerio clone BF71-1N11, *** SEQUENCING IN PROGRESS ***, in
            unordered pieces.
ACCESSION   AL627261
VERSION     AL627261.4  GI:17974055
KEYWORDS    HTG; HTGS_PHASE1; HTGS_DRAFT; HTGS_FULLTOP.
SOURCE      zebrafish.
  ORGANISM  Danio rerio
            Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
            Actinopterygii; Neopterygii; Teleostei; Euteleostei; Ostariophysi;
            Cypriniformes; Cyprinidae; Danio.
REFERENCE   1  (sites)
  AUTHORS   Burton,Z.
  TITLE     Direct Submission
  JOURNAL   Submitted (18-DEC-2001) Wellcome Trust Sanger Institute, Hinxton,
            Cambridgeshire, CB10 1SA, UK. E-mail enquiries:
            humquery@sanger.ac.uk Clone requests: clonerequest@sanger.ac.uk
COMMENT     On Dec 20, 2001 this sequence version replaced gi:15508354.
            -------------- Genome Center
            Center: Wellcome Trust Sanger Institute
            Center code: SC
            Web site: http://www.sanger.ac.uk
            Contact: humquery@sanger.ac.uk
            -------------- Project Information
            Center project name: BZUN11
            -------------- Summary Statistics
            Assembly program: XCAP4; version 4.5
            Sequencing vector: plasmid; L08752; 100% of reads
            Chemistry: Dye-terminator Big Dye; 100% of reads
            Consensus quality: 145782 bases at least Q40
            Consensus quality: 146187 bases at least Q30
            Consensus quality: 146573 bases at least Q20
            Insert size: 146977; sum-of-contigs
            Insert size: 133003; 18.5% error; agarose-fp
            Quality coverage: 9.18x in Q20 bases; sum-of-contigs Quality
            coverage: 10.24x in Q20 bases; agarose-fp
            --------------
            * NOTE: This is a 'working draft' sequence.
            * This record will be updated with the finished sequence
            * as soon as it is available and the accession number will
            * be preserved.
FEATURES             Location/Qualifiers
     source          1..147777
                     /organism="Danio rerio"
                     /db_xref="taxon:7955"
                     /clone="BF71-1N11"
                     /clone_lib="RPCI-71"
     misc_feature    1..11250
                     /note="assembly_fragment:01799
                     fragment_chain:1
                     clone_end:SP6
                     vector_side:left"
     misc_feature    11191..28894
                     /note="assembly_fragment:02192
                     fragment_chain:1"
     misc_feature    28995..31208
                     /note="assembly_fragment:02277
                     fragment_chain:1"
     misc_feature    31309..44590
                     /note="assembly_fragment:01705
                     fragment_chain:1"
     misc_feature    44691..50341
                     /note="assembly_fragment:02974
                     fragment_chain:1"
     misc_feature    50442..77356
                     /note="assembly_fragment:02335
                     fragment_chain:1"
     misc_feature    77457..85291
                     /note="assembly_fragment:03275
                     fragment_chain:1"
     misc_feature    85392..109028
                     /note="assembly_fragment:01218
                     fragment_chain:1"
     misc_feature    109129..147777
                     /note="assembly_fragment:01091
                     fragment_chain:1
                     clone_end:T7
                     vector_side:right"
BASE COUNT    45528 a   26772 c   26968 g   47709 t     800 others
ORIGIN


  Query Match             95.0%;  Score 19;  DB 2;  Length 147777;
  Best Local Similarity   100.0%;  Pred. No. 19;
  Matches   19;  Conservative    0;  Mismatches    0;  Indels    0;  Gaps    0;

Qy       2 ctgatatttctggtgggaaa 20
           |||||||||||||||||||
Db   99420 CTGATATTCTGGTGGGAAA 99402


RESULT  10
AC026534/c
LOCUS       AC026534               66227 bp    DNA     linear   HTG 22-MAR-2000
DEFINITION  Homo sapiens chromosome 17 clone RP11-362L21 map 17, LOW-PASS
            SEQUENCE SAMPLING.
ACCESSION   AC026534
VERSION     AC026534.1  GI:7283233
KEYWORDS    HTG; HTGS_PHASE0.
SOURCE      human.
  ORGANISM  Homo sapiens
            Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
            Mammalia; Eutheria; Primates; Catarrhini; Hominidae; Homo.
REFERENCE   1  (bases 1 to 66227)
  AUTHORS   Birren,B., Linton,L., Nusbaum,C. and Lander,E.
  TITLE     Homo sapiens chromosome 17, clone RP11-362L21
  JOURNAL   Unpublished
REFERENCE   2  (bases 1 to 66227)
  AUTHORS   Birren,B., Linton,L., Nusbaum,C., Lander,E., Abraham,H., Allen,N.,
            Anderson,S., Baldwin,J., Barna,N., Bastien,V., Beda,F.,
            Boguslavkiy,L., Bouhgalter,B., Brown,A., Burkett,G.,
            Campagiano,A., Castle,A., Choepel,Y., Colangelo,M., Collins,S.,
            Collymore,A., Cooke,P., DeArellano,K., Dewar,K., Diaz,J.S.,
            Dodge,S., Domino,M., Doyle,M., Ferreira,P., FitzHugh,W., Gage,D.,
            Galagan,J., Gardyna,S., Ginde,S., Goyette,M., Graham,L.,
            Grand-Pierre,N., Grant,G., Hagos,B., Heaford,A., Horton,L.,
            Howland,J.C., Iliev,I., Johnson,R., Jones,C., Kann,L., Karatas,A.,
            Klein,J., LaRocque,K., Lamazares,R., Landers,T., Lehoczky,J.,
            Levine,R., Lieu,C., Liu,G., Locke,K., Macdonald,P., Marquis,N.,
            McCarthy,M., McEwan,P., McGurk,A., McKernan,K., McPheeters,R.,
            Meldrim,J., Meneus,L., Mihova,T., Miranda,C., Mlenga,V., Morrow,J.,
            Murphy,T., Naylor,J., Norman,C.H., O'Connor,T., O'Donnell,P.,
            O'Neil,D., Olivar,T.M., Oliver,J., Peterson,K., Pierre,N.,
            Pisani,C., Pollara,V., Raymond,C., Riley,R., Rogov,P., Rothman,D.,
            Roy,A., Santos,R., Schauer,S., Severy,P., Spencer,B.,
            Stange-Thomann,N., Stojanovic,N., Subramanian,A., Talamas,J.,
            Tesfaye,S., Theodore,J., Tirrell,A., Travers,M., Trigilio,J.,
            Vassiliev,H., Viel,R., Vo,A., Wilson,B., Wu,X., Wyman,D., Ye,W.J.,
            Young,G., Zainoun,J., Zimmer,A. and Zody,M.
  TITLE     Direct Submission
  JOURNAL   Submitted (22-MAR-2000) Whitehead Institute/MIT Center for Genome
            Research, 320 Charles Street, Cambridge, MA 02141, USA
COMMENT     All repeats were identified using RepeatMasker:
```

```
     *     49478    52118: contig of 2641 bp in length
     *     52119    52218: gap of unknown length
     *     52219    54750: contig of 2532 bp in length
     *     54751    54850: gap of unknown length
     *     54851    57506: contig of 2656 bp in length
     *     57507    57606: gap of unknown length
     *     57607    61230: contig of 3624 bp in length
     *     61231    61330: gap of unknown length
     *     61331    63854: contig of 2524 bp in length
     *     63855    63954: gap of unknown length
     *     63955    69621: contig of 3667 bp in length
     *     69622    69721: gap of unknown length
     *     69722    71798: contig of 2077 bp in length
     *     71799    71898: gap of unknown length
     *     71899    75809: contig of 3911 bp in length
     *     75810    75909: gap of unknown length
     *     75910    80189: contig of 4280 bp in length
     *     80190    80289: gap of unknown length
     *     80290    84857: contig of 4568 bp in length
     *     84858    84957: gap of unknown length
     *     84958    88826: contig of 3869 bp in length
     *     88827    88926: gap of unknown length
     *     88927    93727: contig of 4801 bp in length
     *     93728    93827: gap of unknown length
     *     93828    97828: contig of 4001 bp in length
     *     97829    97928: gap of unknown length
     *     97929   103704: contig of 5776 bp in length
     *    103705   103804: gap of unknown length
     *    103805   109139: contig of 5335 bp in length
     *    109140   109239: gap of unknown length
     *    109240   114126: contig of 4887 bp in length
     *    114127   114226: gap of unknown length
     *    114227   119186: contig of 4960 bp in length
     *    119187   119286: gap of unknown length
     *    119287   124397: contig of 5111 bp in length
     *    124398   124497: gap of unknown length
     *    124498   131793: contig of 7296 bp in length
     *    131794   131893: gap of unknown length
     *    131894   138694: contig of 6801 bp in length
     *    138695   138794: gap of unknown length
     *    138795   145465: contig of 6671 bp in length
FEATURES             Location/Qualifiers
     source          1..145465
                     /organism="Homo sapiens"
                     /db_xref="taxon:9606"
                     /chromosome="3"
                     /map="3p"
                     /clone="RP11-348L20"
     misc_feature    1..1322
                     /note="assembly_name:Contig31"
     misc_feature    1423..2860
                     /note="assembly_name:Contig12"
     misc_feature    2961..4011
                     /note="assembly_name:Contig13"
     misc_feature    4112..5286
                     /note="assembly_name:Contig18"
     misc_feature    5387..7432
                     /note="assembly_name:Contig20"
     misc_feature    7533..8990
                     /note="assembly_name:Contig22"
     misc_feature    9091..10431
                     /note="assembly_name:Contig23"
     misc_feature    10532..12239
                     /note="assembly_name:Contig24"
     misc_feature    12240..13782
                     /note="assembly_name:Contig26"
     misc_feature    13883..15005
                     /note="assembly_name:Contig29"
     misc_feature    15106..16276
                     /note="assembly_name:Contig30"
     misc_feature    16377..18085
                     /note="assembly_name:Contig31"
     misc_feature    18186..19767
                     /note="assembly_name:Contig32"
     misc_feature    19868..21770
                     /note="assembly_name:Contig34"
     misc_feature    21871..24168
                     /note="assembly_name:Contig35"
     misc_feature    24269..26094
                     /note="assembly_name:Contig36"
     misc_feature    26195..29446
                     /note="assembly_name:Contig37"
     misc_feature    29547..32263

  Query Match          100.0%;  Score 20;  DB 2;  Length 145465;
  Best Local Similarity  100.0%;  Pred. No. 5.5;
  Matches   20;  Conservative    0;  Mismatches    0;  Indels    0;  Gaps    0;

Qy       1 gctgatattctggtgggaaaa 20
           ||||||||||||||||||||
Db   47641 GCTGATATTCTGGTGGGAAA 47661


RESULT   8
AC104653/c
LOCUS       AC104653              185276 bp    DNA     linear   HTG 21-JAN-2002
DEFINITION  Homo sapiens chromosome 2 clone RP11-295D2, *** SEQUENCING IN
            PROGRESS ***, 35 unordered pieces.
ACCESSION   AC104653
VERSION     AC104653.2  GI:18252751
KEYWORDS    HTG; HTGS_PHASE1.
SOURCE      human.
  ORGANISM  Homo sapiens
            Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
            Mammalia; Eutheria; Primates; Catarrhini; Hominidae; Homo.
REFERENCE   1  (bases 1 to 185276)
  AUTHORS   Waterston,R.H.
  TITLE     The sequence of Homo sapiens clone
  JOURNAL   Unpublished
REFERENCE   2  (bases 1 to 185276)
  AUTHORS   Waterston,R.H.
  TITLE     Direct Submission
  JOURNAL   Submitted (18-DEC-2001) Genome Sequencing Center, Washington
            University School of Medicine, 4444 Forest Park Parkway, St. Louis,
            MO 63108, USA
COMMENT     On Jan 21, 2002 this sequence version replaced gi:17921251.

            ---------------- Genome Center ----------------
            Center: Washington University Genome Sequencing Center
            Center code: WUGSC
            Web site:http://genome.wustl.edu/gsc/index.shtml
            ---------------- Project Information ----------------
            Center project name: H_NH0295D02
            ---------------- Summary Statistics ----------------
            Sequencing vector: M13; 69%
            Sequencing vector: plasmid; 31%
            Chemistry: Dye-primer ET; 0% of reads
            Chemistry: Dye-terminator Big Dye; 100% of reads
            Assembly program: Phrap; version 0.990319
            Consensus quality: 173270 bases at least Q40
            Consensus quality: 175899 bases at least Q30
            Consensus quality: 177845 bases at least Q20
            ----------------------------------------------------

            * NOTE: This is a 'working draft' sequence. It currently
            * consists of 35 contigs. The true order of the pieces
            * is not known and their order in this sequence record is
            * arbitrary. Gaps between the contigs are represented as
            * runs of N, but the exact sizes of the gaps are unknown.
            * This record will be updated with the finished sequence
            * as soon as it is available and the accession number will
            * be preserved.
            *       1     1390: contig of 1390 bp in length
            *    1391     1490: gap of unknown length
            *    1491     3004: contig of 1514 bp in length
            *    3005     3104: gap of unknown length
            *    3105     4782: contig of 1678 bp in length
```

```
COMMENT     ----------

            NOTICE:  This sequence may not represent the entire insert of this
            clone.  It may be shorter because we only sequence overlapping
            clone sections once, or longer because we provide a small overlap
            between neighboring data submissions.

            This sequence was finished as follows unless otherwise noted:
            all regions were double stranded, sequenced with an alternate
            chemistry, or covered by high quality data (i.e., phred quality >=
            30); an attempt was made to resolve all sequencing problems, such
            as compressions and repeats; all regions were covered by sequence
            from more than one subclone; and the assembly was confirmed by
            restriction digest.

            MAPPING INFORMATION:
            Mapping information for this clone was provided by Dr. John D.
            McPherson, Department of Genetics, Washington University, St. Louis
            MO.  For additional information about the map position of this
            sequence, see http://genome.wustl.edu/gsc

            SOURCE INFORMATION:
            The RPCI-11 human BAC library was made from the blood of one male
            donor, as described by Osoegawa,K., Woon,P.Y., Zhao,B., Frengen,E.,
            Tateno,M., Catanese,J.J. and de Jong,P.J. (1998) An improved
            approach for construction of bacterial artificial chromosome
            libraries.  Genomics 52:1-8.  The clone may be obtained either from
            Research Genetics, Inc. (http://www.resgen.com) or Pieter de Jong
            and coworkers at the Roswell Park Cancer Institute
            (http://bacpac.med.buffalo.edu)
            VECTOR:  pBACe3.6
            NEIGHBORING SEQUENCE INFORMATION:
            The clone sequenced to the left is AL078621, 2000 bp overlap.
            Actual end is at base position 142432 of RP11-432D15.

            There is an unresolved region from 107145 to 107172. Single plasmid
            region exists between 107078 and 107171.
FEATURES                    Location/Qualifiers
     source                 1..142432
                            /organism="Homo sapiens"
                            /db_xref="taxon:9606"
                            /chromosome="2"
                            /map="2"
                            /clone="RP11-432C15"
                            /clone_lib="RPCI-11"
     repeat_region          250..545
                            /rpt_family="Alu"
     repeat_region          816..867
                            /rpt_family="L2"
     repeat_region          1020..1313
                            /rpt_family="Alu"
     repeat_region          1433..1727
                            /rpt_family="Alu"
     misc_feature           1970..2630
                            /note="similar to EST AW070632 (NID:g6025630) xa04d07.x1"
     misc_feature           2822..2958
                            /note="similar to EST BG724168 (NID:g14003355)"
     repeat_region          4028..4424
                            /rpt_family="ERV1"
     repeat_region          4751..4777
                            /rpt_family="(TTTTTG)n"
     repeat_region          6521..6686
                            /rpt_family="MaLR"
     repeat_region          6687..6887
                            /rpt_family="MaLR"
     repeat_region          6888..7234
                            /rpt_family="ERV1"
     repeat_region          7235..7467
                            /rpt_family="MaLR"
     repeat_region          7468..7856
                            /rpt_family="MaLR"
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                            /rpt_family="MIR"
     repeat_region          10123..10214
                            /rpt_family="MIR"
     repeat_region          10272..10430
                            /rpt_family="MIR"
     repeat_region          11094..11309
                            /rpt_family="MaLR"
     repeat_region          11278..11411
                            /rpt_family="MaLR"
     repeat_region          12126..12234
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     repeat_region          12315..12623
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     repeat_region          13546..15375
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     repeat_region          15358..15413
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     repeat_region          15414..15700
                            /rpt_family="Alu"
     repeat_region          15701..16452
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     repeat_region          15901..15966
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     repeat_region          16455..17157
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                            /rpt_family="L1"
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ORIGIN

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  Best Local Similarity  100.0%;  Pred. No. 9.1;
  Matches   20;  Conservative    0;  Mismatches    0;  Indels    0;  Gaps    0;

Qy      1 gctgatattctggtgggaaa 20
          ||||||||||||||||||||
Db      1 GCTGATATTCTGGTGGGAAA 20


RESULT  2
AX052810
LOCUS       AX052810                 20 bp    DNA     linear   PAT 12-JAN-2001
DEFINITION  Sequence 20 from Patent WO0071753.
ACCESSION   AX052810
VERSION     AX052810.1  GI:12226967
KEYWORDS    .
SOURCE      synthetic construct.
  ORGANISM  synthetic construct
            artificial sequence.
REFERENCE   1  (bases 1 to 20)
  AUTHORS   Kornman,K.S., Duff,G.W., Crossman,D.C., Francis,S.E. and
            Stephenson,K.
  TITLE     Diagnostics and therapeutics for restenosis
  JOURNAL   Patent: WO 0071753-A 20 30-NOV-2000;
            Interleukin Genetics, Inc. (US)
FEATURES             Location/Qualifiers
     source          1..20
                     /organism="synthetic construct"
                     /db_xref="taxon:32630"
                     /note="primer"
BASE COUNT         5 a      2 c      7 g      6 t
ORIGIN

  Query Match           100.0%;  Score 20;  DB 6;  Length 20;
  Best Local Similarity  100.0%;  Pred. No. 9.1;
  Matches   20;  Conservative    0;  Mismatches    0;  Indels    0;  Gaps    0;

Qy      1 gctgatattctggtgggaaa 20
          ||||||||||||||||||||
Db      1 GCTGATATTCTGGTGGGAAA 20


RESULT  3
AX067270
LOCUS       AX067270                 20 bp    DNA     linear   PAT 24-JAN-2001
DEFINITION  Sequence 6 from Patent WO0100880.
ACCESSION   AX067270
VERSION     AX067270.1  GI:12544894
KEYWORDS    .
SOURCE      synthetic construct.
  ORGANISM  synthetic construct
            artificial sequence.
REFERENCE   1  (bases 1 to 20)
  AUTHORS   Duff,G.W., Cox,A., Camp,N.J. and di Giovine,F.S.
  TITLE     Diagnostics and therapeutics for diseases associated with an il-1
            inflammatory haplotype
  JOURNAL   Patent: WO 0100880-A 6 04-JAN-2001;
            Interleukin Genetics, Inc. (US)
FEATURES             Location/Qualifiers
     source          1..20
                     /organism="synthetic construct"
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                     /note="primer"
BASE COUNT         5 a      2 c      7 g      6 t
ORIGIN

  Query Match           100.0%;  Score 20;  DB 6;  Length 20;
  Best Local Similarity  100.0%;  Pred. No. 9.1;
  Matches   20;  Conservative    0;  Mismatches    0;  Indels    0;  Gaps    0;

Qy      1 gctgatattctggtgggaaa 20
          ||||||||||||||||||||
Db      1 GCTGATATTCTGGTGGGAAA 20


RESULT  4
HS087XA1
LOCUS       HS087XA1                 213 bp    DNA     linear   PRI 28-NOV-1994
DEFINITION  H. sapiens (D2S121) DNA segment containing (CA) repeat; clone
            AFM087xa1; single read.
ACCESSION   Z16545
VERSION     Z16545.1  GI:23080
KEYWORDS    CA repeat; dinucleotide repeat; GT repeat; microsatellite DNA;
            microsatellite marker; repeat polymorphism.
SOURCE      human.
  ORGANISM  Homo sapiens
            Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
            Mammalia; Eutheria; Primates; Catarrhini; Hominidae; Homo.
REFERENCE   1  (bases 1 to 213)
  AUTHORS   Weissenbach,J.
  TITLE     Direct Submission
  JOURNAL   Submitted (05-OCT-1992) Genethon, B.P. 60, 91002 Evry Cedex France.
            E-mail: Jean.Weissenbach@genethon.fr
REFERENCE   2  (bases 1 to 213)
  AUTHORS   Weissenbach,J., Gyapay,G., Dib,C., Vignal,A., Morissette,J.,
            Millasseau,P., Vaysseix,G. and Lathrop,M.
  TITLE     A second-generation linkage map of the human genome
  JOURNAL   Nature 359 (6398), 794-801 (1992)
  MEDLINE   93063290
REFERENCE   3  (bases 1 to 213)
  AUTHORS   Gyapay,G., Morissette,J., Vignal,A., Dib,C., Fizames,C.,
            Millasseau,P., Marc,S., Bernardi,G., Lathrop,M. and Weissenbach,J.
  TITLE     The 1993-94 Genethon human genetic linkage map
  JOURNAL   Nat. Genet. 7 (2 Spec No), 246-339 (1994)
  MEDLINE   95004593
COMMENT     cloning vector is M13mp18;
            full automatic.
FEATURES             Location/Qualifiers
     source          1..213
                     /organism="Homo sapiens"
                     /db_xref="taxon:9606"
                     /chromosome="2"
                     /cell_line="CEPH 134702"
                     /clone_lib="genomic DNA"
BASE COUNT        73 a     49 c     42 g     49 t
ORIGIN

  Query Match           100.0%;  Score 20;  DB 9;  Length 213;
  Best Local Similarity  100.0%;  Pred. No. 7.9;
  Matches   20;  Conservative    0;  Mismatches    0;  Indels    0;  Gaps    0;

Qy      1 gctgatattctggtgggaaa 20
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Db      2 GCTGATATTCTGGTGGGAAA 21


RESULT  5
AC069268
LOCUS       AC069268              125685 bp    DNA     linear   HTG 23-MAY-2000
DEFINITION  Homo sapiens chromosome 3p clone RP11-348I20, WORKING DRAFT
            SEQUENCE, 31 unordered pieces.
ACCESSION   AC069268
VERSION     AC069268.1  GI:8039671
KEYWORDS    HTG; HTGS_PHASE1; HTGS_DRAFT.
SOURCE      human.
  ORGANISM  Homo sapiens
            Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
            Mammalia; Eutheria; Primates; Catarrhini; Hominidae; Homo.
REFERENCE   1  (bases 1 to 125685)
  AUTHORS   Wang,J., Hu,S., Dong,W., Wang,J., Zhang,Y., Zhang,H., Liu,B.,
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